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PRACTICAL RECOGNITION AND TREATMENT OF CARDIAC ARRYTHMIAS

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DURING the oral presentation of this paper, slides of electrocardiograms illustrating the various cardiac arrhythmias were shown. Since this is not feasible in a written paper, the section on diagnosis is somewhat abbreviated and more space is given to treatment. I am eschewing any textbook version of this subject; instead, I am relying on practical experience, teaching and research in this field, particularly in the past decade. I might say that the subject of cardiac arrhythmias has been a hobby of mine and I am both honored and delighted to discuss it with you. I am aiming at a practical exposition of this problem bearing in mind what one can do in the office or in the home.

The arrhythmias to be discussed are as follows:

1. Sinus tachycardia
2. Sinus bradycardia
3. Sinus arrhythmia
4. Premature systoles
   a. Auricular
   b. Nodal
   c. Ventricular
5. Supraventricular tachycardia, which includes:
   a. Auricular tachycardia
   b. Nodal tachycardia
6. Auricular fibrillation
7. Auricular flutter
8. Heart block
9. Ventricular tachycardia
10. Ventricular flutter and fibrillation

It is worth recalling that the generating impulse which initiates and maintains cardiac contraction arises in the sino-auricular (s-a) node, which is located in the right auricle near the opening of the coronary sinus. It then spreads to the auricular muscle and reaches the auriculoventricular (a-v) node, from which it spreads to the common bundle of His located in the upper one-third of the interventricular septum. The impulse then divides into the right and left bundle in the right and left ventricles beneath the endocardium and finally terminates in the myocardium as a fine arborization termed the Purkinje fibers. These specialized areas, such as the s-a and the a-v node consist of specialized muscle tissue, not nerve tissue. They have the unique property of maintained or constantly functioning irritability and rhythmicity. All portions of the heart muscles have irritability and rhythmicity but to a lesser degree than that described for the above specialized areas.

1. Sinus tachycardia:

This is diagnosed when the impulse originates from the s-a node at a rate of 100-180. Heart rates above 180 are usually considered to be outside the s-a node because the rhythmicity of the latter usually does not exceed 180 beats per minute. When in doubt, of course, an electrocardiogram (EKG) should be taken; the presence of a P wave of normal contour signifies that the rhythm is originating from the s-a node. The best treatment is to find the cause, and to eliminate that, for there is no drug which specifically and solely slows down the s-a node. Digitalis, mecholyl, and the veratrum alkaloids (1) will stimulate the vagus or cardioinhibitory nerve but these drugs are not used solely for sinus tachycardia.

2. Sinus bradycardia:

This is diagnosed when the impulse originates from the s-a node at a rate below 60 but usually not below 30 beats per minute. This is most often a perfectly normal finding and requires no treatment at all. A regular pulse around 30 beats

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per minute should arouse the suspicion of heart block; an EKG will elucidate this.

3. Sinus arrhythmia:

This consists of two types: phasic (varying with respiration) and non-phasic (not varying with respiration). It is commonly found in children and young adults and when marked it may be clinically confused with abnormal and ectopic (that is, located outside the normal pacemakers) rhythms, such as auricular fibrillation; indeed, at times I have not been able to differentiate phasic sinus arrhythmia from auricular fibrillation except by the EKG. Usually, however, the presence of phasic sinus arrhythmia may be ascertained by having the patient hold the breath; then the irregularity of the heart beat or pulse disappears and resumes again after breathing. This is important because, aside from presenting clinical confusion, it is a perfectly normal rhythm and requires no treatment. It is caused by variations in the physiological tone or activity of the vagus nerve and commonly disappears in adult life. One other important point must be mentioned: an electrocardiogram of phasic sinus arrhythmia may not infrequently show variations in the height of the P wave (termed wandering pacemaker within the s-a node) and this may lead the unwary interpreter to diagnose an abnormal rhythm, which it definitely is not.

4. Premature systoles:

Except for normal sinus rhythm, premature beats are the most common type of arrhythmia. The patient complains of a sudden awareness of the heart beat or cardiac consciousness, a sudden jumping or flopping of the heart and a pause which excites a dread as to when the next beat will start again. It is usually not possible clinically to determine whether the beat arises in the auricle, a-v node or ventricle. In the electrocardiogram these have specific characteristics. The auricular premature beat is always preceded by a P wave, the P-R interval is greater than 0.12 seconds and the QRS complex is not wider than 0.11 seconds; in addition, the premature QRS complex resembles the normal QRS complex. The nodal premature beat is usually not preceded by a P wave although it may be followed by one (a retrograde P wave), the QRS is of normal width and resembles the normal or sinus QRS complexes. Nodal premature beats are uncommon. The ventricular extrasystole is never preceded by a P wave, the QRS is wider than 0.11 seconds and is bizarre in appearance, that is, looks quite different from the normal or sinus QRS complexes. This is important to know for runs of premature auricular or ventricular systols can, under certain conditions of myocardial anoxemia, give rise to auricular tachycardia and fibrillation and ventricular tachycardia and fibrillation respectively.

When premature beats do not cause symptoms and myocardial infarction is not present, no treatment is required. When symptoms described above are present and the patient is disturbed I have found the following therapeutic program most helpful:

(1) Reassurance: This should be forceful, convincing and based on a thorough examination. Patients will want to know that they do not cause heart disease or sudden death and that the heart always beats again. Extra systoles may occur with heart disease but also with peptic ulcer and cholecystitis or other diseases.

(2) Correct any underlying condition, notably anemia, excessive coffee, tobacco or alcohol, fatigue and apprehension.

(3) Sedation to diminish cardiac consciousness and apprehension: Phenobarbital and bromides are quite adequate. It is best to prescribe them just before the patient has his symptoms such as late in the afternoon, rather than routinely.

(4) Treatment which the patient can perform: pressing firmly on the eyeballs until they hurt, pulling out the tongue to the point of gagging, holding the head between the knees, or an ice bag on the chest. In the main, I have not had too much success with these maneuvers but they are worth passing on to the patient.

(5) Specific treatment:
- (a) quinidine
- (b) papaverine
- (c) procaine amide or oral pronestyl (Squibb) (best in ventricular premature beats)
- (d) potassium salts
- (e) digitalis

Digitalis is placed last because in most patients the other drugs produce the desired therapeutic effect. Any of them may be combined with phenobarbital or bromides. The matter of dosage is considered at the end of this presentation in order to avoid repetition.

5. Supraventricular tachycardia:

A run of three or more premature beats, originating from the auricle or the a-v node constitutes technically a paroxysmal tachycardia. If the P wave is seen preceding the QRS complex

then a definite diagnosis of paroxysmal auricular tachycardia may be made. However, when the rate is fast, e.g., over 180 beats per minute approximately, the P and T waves often cannot be identified. Hence, auricular and nodal tachycardias are grouped under the heading of supraventricular. On auscultation, the rate is fast, that is, 180 beats per minute or more, precisely regular, its rate does not vary with respiration; if the rate is slowed by carotid sinus pressure, it returns rapidly and regularly to its pre-carotid sinus pressure rate. Its clinical distinction from ventricular tachycardia is listed under the latter. I have found this clinical dictum helpful: a rapid regular rate is more common in the normal heart, or the diseased heart without myocardial infarction, and in that case the arrhythmia is presumptively supraventricular.

The treatment may be divided in to therapy during the attack and therapy between the attacks.

Treatment during the attack:

(1) Bed rest: this is not obligatory but the patient will often take to bed anyway.

(2) Over the telephone one may advise eyeball pressure, pulling out the tongue as discussed above. An ice bag on the chest is useful for cold usually diminishes irritability and rhythmicity in the myocardium. Also worth trying is the Valsalva maneuver, namely, taking a deep inspiration, holding the breath and exhaling forcefully.

(3) Sedation: giving barbiturates or morphine sulphate is useful for two reasons:
   (a) Apprehension and cardiac consciousness are decreased.
   (b) Both these agents sensitize the vagus nerve, permitting subsequent carotid sinus pressure to be more effective. This is a common experience and of great importance.

(4) Carotid sinus pressure: This area can best be located by following the carotid artery craniad until its bifurcation is reached. One not infrequently hears the resident physician say that this maneuver is unsuccessful when actually he was not pressing on the carotid sinus. I have found carotid sinus pressure much superior to massage near the carotid sinus. Since hemiplegia has been reported (2) from bilateral carotid sinus pressure, unilateral pressure, trying one side at a time, is recommended. However, I have at times, especially in the younger age groups, stopped the tachycardia with bilateral pressure after unilateral pressure proved ineffective. In youngsters, the carotid sinus is much less sensitive than in adults, so that premedication or sensitization of the vagus nerve with the medication mentioned above, or, in addition using prostigmine, is essential.

(5) Digitalis: For speed and effectiveness of action, digitalis is often unsurpassed, particularly the newer purified glycosides like digoxin and Lanatocide C (Cedilanid). The same result is achieved orally or intravenously, the only difference being, of course, speed of action. Since the patient with supraventricular tachycardia is grateful for a quick termination, probably intravenous digitalization is desirable. A full digitalizing dose need not be used; I usually give half a full digitalizing dose and then wait fifteen to thirty minutes or else apply carotid sinus pressure again, since digitalis also produces vagal sensitization. With the older digitalis leaf preparations, extra digitalis was not given until two weeks had elapsed since its discontinuance, allowing that period of time for excretion. However, the purified glycosides are excreted in from one to four day and then additional digitalis (purified) may then be given.

(6) Quinidine: This may be given in doses which are spread out or concentrated, depending on the speed of action desired. They may be spread out in doses of 3-6 grains every four hours day and night until toxic symptoms occur; or, in doses of six grains every two hours for five doses, every day, the physician always watching for toxicity.

Many cardiologists prefer to administer digitalis before quinidine, since the former will slow the rate even if it does not stop the tachycardia. Stubborn supraventricular tachycardias, which have lasted for days or weeks, are more often terminated by digitalis than by quinidine (3). It is entirely feasible to give quinidine after the patient has received digitalis.

(7) Neosynephrine: Youmans (4) has recently described excellent and rapid re-

sults from the use of 0.3-0.1 cc. of 1% Neosynephrine solution given intravenously. The average dose is from 0.3-0.5 cc. The aim of therapy is to raise the blood pressure, provoke cardioinhibitory reflexes via the aortic arch and carotid sinus and thereby slow or terminate the tachycardia. Cessation of the arrhythmia has occurred within forty-five seconds of injection. I can confirm his results in a smaller number of cases. Probably the only contraindication is initial severe hypertension. With average doses, no toxic effects have occurred.

(8) Mecholyl: This is a rapidly acting drug with powerful side effects. I never give this drug without having available at least grains 1/100 of atropine in a syringe, with a patent needle and a tourniquet on the arm, with a distended vein available for immediate injection of atropine should the blood pressure and pulse drop excessively. As an added precaution, probably the electrocardiogram machine should be attached to the patient. Atropine will quickly counteract the side effects of mecholyl but it must be available. Recently (5) the intranasal administration of mecholyl has been described; the drug is freshly dissolved, a cotton applicator is dipped therein and the applicator placed in one nostril for several seconds. The side effects are less powerful and the results are promising.

(9) Magnesium sulphate: This is given slowly by vein. It is not often used but Scherf (6) has described good results. During administration, it is wise to listen at the apex or check the attached direct writing electrocardiograph machine for toxic arrhythmias, such as frequent ventricular premature systoles, have been described.

(10) Syrup of Ipecac: An old remedy particularly valuable in children, although the vomiting induced may not be appreciated by the patient. It has a wide margin of safety.

All the above drugs and maneuvers act by stimulating the vagus nerve or depressing irritability in the auricular myocardium or both.

Treatment between the attacks:

(1) If rare, no treatment is needed.

(2) If frequent, one may use either quinidine or digitalis often combined with daily sedation (barbiturates or bromides). Despite these drugs, episodes of supraventricular tachycardia may still recur.

6. Auricular Fibrillation:

A completely irregular rhythm is most likely auricular fibrillation; the rate usually varies from 100-200 beats per minute. At the onset or before treatment, the ventricular rate is often faster than the pulse rate constituting a pulse deficit. The irregularity of this arrhythmia is simulated only by frequent premature systoles from different myocardial foci or by marked sinus arrhythmia and the distinction is then made by the electrocardiogram.

Since only two drugs are used in its treatment (including both the paroxysmal and chronic types) it seems to be advantageous to discuss choice of therapy, that is, when digitalis or quinidine should be used:

A. Digitalis should be used:
   a When congestive heart failure is caused by the rapid ventricular rate.
   b When slowing of a rapid ventricular rate is desired rather than abolition of the arrhythmia.
   c When quinidine has been tried and found ineffective.

B. Quinidine should be used:
   a When abolition of the arrythmia is desired, especially after digitalis has slowed the rate without terminating the auricular fibrillation.
   b In a recent paroxysm of short duration, particularly when the heart is normal.
   c When it complicates myocardial infarction, following anaesthesia, after thyroid surgery or febrile disease, and especially when in these circumstances the paroxysmal auricular fibrillation does not stop spontaneously in a few days.

Patients who have mitral stenosis and auricular fibrillation for some years should not be converted to normal sinus rhythm with quinidine (although there is contrary opinion (7), because they usually revert to auricular fibrillation. On the other hand, patients with mitral stenosis—and they are most apt to develop that arrhythmia—who have auricular fibrillation for the first time should be digitalized to slow the rate and then given quinidine. If the patient does not fibrillate again, he is spared taking digitalis for years to come. If he continues to have auricular fibrillation, then he will have to take digitalis.

Whenever congestive heart failure is present

due to the auricular fibrillation, or any other cause, then digitalis is given first for two reasons: decompensation alone will accelerate the heart rate (probably because of the Bainbridge reflex) and quinidine is more effective when the ventricular rate is slow, that is, less than 100 beats per minute.

7. Auricular Flutter:

This is probably the most missed arrhythmia. It can masquerade as sinus tachycardia or supraventricular tachycardia if every auricular beat is conducted to the ventricle (1:1 conduction) or even if only every second beat is conducted to the ventricle (2:1 conduction). It can mimic auricular fibrillation if the auricular beats are conducted to ventricle at irregular rates, that is 1:1, 3:1, 7:1, and so on. Even in the electrocardiogram, the flutter waves may not be detected unless one is suspicious. A good clinical rule is that any arrhythmia, which does not respond to its customarily appropriate therapy should be suspected of being auricular flutter. One can obtain much diagnostic help by taking a precordial lead electrocardiogram both before and after carotid sinus pressure. The bradycardia or cardiac stoppage temporarily produced by carotid pressure will make the flutter waves clear in the EKG.

The treatment of auricular flutter may be outlined thus:

A. Digitalize first. Roughly 50-70% of flutter patients will be converted to auricular fibrillation. When the latter occurs, stop digitalis promptly and about 50% of such patients will spontaneously change back to normal sinus rhythm. This necessitates slowly increasing doses of digitalis and frequent observation each day in order not to miss the time when flutter is changed to fibrillation. Should more digitalis than is needed to effect this change be given, then the patient may remain permanently in auricular fibrillation.

B. Give quinidine to those patients who do not change from flutter to fibrillation with digitalis, or do not revert from fibrillation to normal sinus rhythm spontaneously. 15-25% approximately of this group will change back to normal rhythm.

The change from auricular fibrillation to normal rhythm without medication usually occurs in from a few hours to a few days.

The following case illustrates some of the points mentioned in the therapy of auricular flutter and fibrillation:

A fifty year old white female with rheumatic heart disease and mitral stenosis, whom I have been following for several years, developed for the first time on April 1, 1950, a rapid and irregular heart rate of 140 beats per minute, dyspnea, mild orthpnea, and basal pulmonary rales. The EKG seemed to show auricular fibrillation but carotid sinus pressure caused a temporary slowing of the rate to disclose (well seen in Lead V2) auricular flutter. This was her first episode of congestive heart failure, very likely precipitated by the arrhythmia. She immediately received four tablets or 1.0 mgs. of digoxin orally on April 1, 1950 and two tablets or 0.50 mgs. on April 2, 1950. Cardiac compensation was restored and the flutter converted to auricular fibrillation on April 3, 1950 at a rate of from 98-108 beats per minute. On April 5, 1950, normal sinus rhythm was restored. However, the patient still had fatigue and mild dyspnea and hence one tablet of digoxin was given daily. On April 10, 1950, auricular flutter reappeared. Since the patient had already been digitalized, she received quinidine grains three (0.1 Grams) every four hours day and night. This was increased to grains six (0.2 Grams) on April 12, 1950 and the auricular flutter stopped on April 13, 1950.

Two or three similar episodes of auricular flutter took place in the ensuing month lasting about one day. Since May 1950 she has been taking one tablet of digoxin and one tablet (grains 3) of quinidine four times a day without any recurrence.

The auricular arrhythmias discussed above have special aspects when they complicate myocardial infarction as was pointed out in a previous paper given before the Arizona Medical Association (8).

8. Heart Block:

This is a less common type of arrhythmia. In the past five years I have seen only two cases of complete heart block. First degree heart block, which is common, consists of a prolongation of the P-R interval beyond the normal of 0.21 seconds and may be caused by infections like rheumatic fever and by drugs. Second degree heart block also has a prolonged P-R interval but some beats are not conducted from the s-a node to the a-v node and are "dropped." Third degree heart block consists of a complete disassociation between the auricle and ventricle, due to disease in the a-v node or high up in the interventricular septum, so that the P waves and QRS complexes have no causal rela-

tionship. In third degree heart block the ventricular rate is usually about thirty beats a minute. This alone is not proof of complete heart block, for blocked out, frequent auricular premature beats may simulate this; hence the diagnosis should be checked by the electrocardiogram.

No specific treatment is needed for first and second degree heart block except to remove the underlying cause whenever possible. Third degree heart block requires treatment during the stage of asystole (Stokes-Adams syndrome) or when ventricular arrhythmias arise. During that time the patient may have fainting, dizziness or syncope. Treatment during the attack consists of either subcutaneous adrenalin or paredrine hydrobromide (9). Oral ephedrine may also be given. Patients who have frequent attacks of syncope should receive either oral ephedrine, or better, oral paredrine daily. The main purpose of treatment is to increase the irritability of the ventricular myocardium so that ventricular ejection of blood to the brain may occur.

9. Ventricular Tachycardia:

This arrhythmia is rare in the normal heart and is found almost entirely in the diseased heart or as a complication of myocardial infarction. However, once the diagnosis is properly made, institution of adequate treatment may be life saving. Ventricular tachycardia is most often confused with supraventricular tachycardia. Ventricular tachycardia differs from supraventricular tachycardia chiefly in these respects: the rate is not precisely regular and may vary some especially during respiration. It is not slowed by carotid sinus pressure. In the electrocardiogram, two notable distinguishing features are present: the width of the QRS complex is greater than normal (0.12 seconds or more) and the time interval between two beats can vary by as much as 0.03 seconds, whereas in supraventricular tachycardia the variation is rarely more than 0.01 seconds. This accounts for the slight irregularity heard during auscultation in ventricular tachycardia. If the patient has bundle branch block prior to the onset of this arrhythmia, the width of the QRS complex is not a useful distinguishing feature. Certain clinical symptoms have been described such as: symptoms of palpitation and dyspnea, substernal dull ache or heavy feeling, signs of peripheral circulatory failure or even congestive heart failure, dizziness and fainting, but I have also seen these symptoms with the onset of supraventricular tachycardia.

Treatment consists of:

(1) Pronestyl (Squibb procaine amide) (10): given intravenously at a rate no faster than 1 cc. (100 mgs.) per minute. Frequent blood pressure recordings should be made for this drug does induce hypotension. It is probably also wise to run electrocardiographic tracings during its administration so that the injection may be ceased when the tachycardia stops. During anaesthesia, this hypotensive side effect is less apt to occur, and hence pronestyl may be administered faster. The drug may also be given orally. This is a new drug and has great promise.

(2) Quinidine: Until the advent of procaine amide, this was the most effective drug available when given in full doses. Unlike pronestyl, it is attended by considerable danger when given intravenously and until recently the intramuscular preparations available were not too satisfactory. It is less expensive than procaine amide.

(3) Morphine: A brief report by Sabathie (11) claims success in stopping this arrhythmia. I have not had an opportunity to use it.

(4) Papaverine: Experimentally (12) this has been found effective but clinically there are too few reports regarding its use. The same may be said for atropine.

(5) Potassium acetate: This is worth remembering but probably more work should be done with it.

(6) Digitalis is usually contraindicated: This is of the utmost importance. As mentioned before, digitalis works successfully in the management of supraventricular tachycardia and hence every effort should be made to properly distinguish the two arrythmias.

10. Ventricular Flutter and Fibrillation:

Even though these are usually fatal arrhythmias, reference has been made to the use of intra-arterial blood transfusion and manual massage of the heart as offering some hope in a previous paper before the Arizona Medical Association (13).

I should like to add a final word regarding quinidine, which should have been mentioned before, namely the threat of embolization. Like others I have reviewed the literature in an extensive series of cases and have found that the danger of emboli being loosened as a result of

quinidine usage in auricular flutter or auricular fibrillation has been over-emphasized. Emboli may occur in these arrhythmias with or without quinidine. Furthermore, the problem is somewhat academic today since heparin or dicumarol may readily be given.

It is to be noted that a completed discussion of this subject has not been presented. Some aspects have been passed over solely in the interests of simplicity.

SUGGESTED OUTLINE OF DOSAGES

The following table is drawn up for the sake of succintness and represents what has been found by experience to be useful and successful. The apothecary system can easily be translated into the metric system on the basis of 1 grain = 0.065 Grams.

1. Quinidine:
   a. 3 grains every 3 hours for 24 hours or longer.
   b. 6 grains every 2 hours for 5 doses for 2-4 days.
   c. 3-6 grains every 3 or 4 hours day and night.

   Method b constitutes rapid quinidine administration. The schedule chosen by the clinican will depend, in part, on the speed of action desired for intramuscular use, quinidine lactate or gluconate may be used. Watch for toxic effects:
   a. gastro-intestinal: nausea, vomiting, diarrhea.
   b. aural: tinnitus or deafness.
   c. myocardial: ectopic beats, intra-ventricular block, ventricular tachycardia.
2. Digitalis:
   a. Cedilanid (Sandoz, Lanatocide C): 8 cc (1. mg) by vein is a full digitalizing dose or 12 tablets (1 tablet=0.5 mgs) in 48 hours. The daily maintenance dose is 1-3 tablets.
   b. Digitoxin (Digitaline Nativelle): 1.0-1.25 mgs is a full digitalizing dose by vein or 5-6 tablets (1 tablet=0.2 mgs) in 24 hours by mouth. The daily maintenance dose is 0.1-0.2 mgs. (1-2 tablets) by mouth.
   c. Digoxin (Burroughs and Wellcome): 0.75-1.0 mgs is a full digitalizing dose or 4-5 tablets (1 tablet=0.25 mgs) in 24 hours by mouth. The daily maintenance dose is 1-2 tablets.

   Watch for toxic effects:
   a. gastro-intestinal: nausea and/or vomiting.
   b. myocardial: premature beats, first degree a-v block.
   c. eyes: colored or colorless spots or vision.
3. Papaverine:
   a. oral: 1½-3 grains 3 or 4 times a day after meals.
   b. intravenous: 1-3 grains slowly.
4. Procaine amide (Pronestyl, Squibb):
   a. oral: 0.25-0.5 Gm (1-2 capsules) every four to six hours.
   b. intravenous: 200-1000 mgs. slowly, not faster than 100 mgs per minute (1 cc= 100 mgs.).
5. Mecholyl:
   10-25 mgs. given subcutaneously. Have atropine ready.
6. Magnesium sulphate:
   10-15 cc of a 25% solution given slowly by vein.
7. Syrup of ipecac:
   Start with 8 cc. If no effect in 30 minutes, repeat same dose. If no effect in 45 minutes, give 16 cc.
8. In heart block (complete):
   a. adrenalin (1:1000 solution): 0.5-1.0 cc subcutaneously.
   b. ephedrine: grains ⅜ orally 3 times a day.
   c. paredrine hydrobromide: 50 mgs orally three times a day or 20 mgs. intramuscularly.
9. Potassium acetate or chloride: 2 Grams three times a day.

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THE IMPORTANCE OF PREOPERATIVE EVALUATION OF THE PATIENT FOR PROPER PREMEDICATION AND CHOICE OF ANESTHESIA

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1. *Importance of the preoperative visit and evaluation of the patient physically and psychologically.*

EVERY rational patient going to surgery has considerable concern regarding the event. He is interested in those who are going to perform surgery and anesthesia. He of course usually knows the surgeon, but he rarely knows who will give him anesthesia. Not infrequently he is far more concerned about the anesthesia than the surgery. He wants to know what is going to be done; how it is going to be done, is he going to be asleep; how much is it going to hurt or distress him; how is he going to feel afterward. Ordinarily the patient will not ask to see the anesthesiologist if he does not appear, because the patient does not want to seem concerned or apprehensive. But all of us have, I am sure, experienced and appreciated the patient's parting remark at the termination of the preoperative visit: "I am certainly glad you came in; my mind is relieved and now I can get a night's sleep and not worry." The visit has in fact been the beginning of the premedication since it tends to lessen apprehension on the part of the patient. Therefore two important things have been accomplished: physician-patient relation has been established, and the confidence of the patient in the physician has been secured.

Equally important, however, is the factual knowledge the anesthesiologist secures which enables him to formulate a basis for a deliberate, objective evaluation of the best anesthetic or combination of anesthetics to be employed. What are some of those facts that are so useful.

(a) History of previous surgery—how the patient reacted to his surgery and anesthesia and medication.

(b) Any specific fears or interdictions regarding any particular anesthesia.

(c) Any known or suspected allergic reactions to previously administered drugs.

(d) History of any respiratory or circulatory abnormalities or peculiarities. How often does the patient say: "I forgot to tell doctor so-and-so, and probably it isn't important, but as a child I had very severe asthma or shortness of breath,"—or some other danger sign to be considered. You know as well as I that it happens all too often.

The history is of course always reviewed before the visit, because a few well-chosen comments indicating prior knowledge of the patient goes a long way in assuring the patient that you are truly interested in him. After the visit notes are made on the patient's chart indicating any additional history and pertinent facts.

(e) A physical examination is performed, particularly of the respiratory and circulatory systems, and variations from those on the ward, if any, noted. It may have been a week or more since the surgeon last saw the patient, and occasionally things happen in a week.

(f) Of particular importance is pre-knowledge of the patient's quantity of hemoglobin, since a transfusion order is much better accomplished the night before than as an emergency in surgery. The presence of abnormal urine findings may modify the choice of anesthesia and may in certain cases require investigation.

From the above considerations it becomes apparent that we may now begin to consider the problems of satisfactory premedication and evaluate the desirability of a variety of suitable anesthetic agents and methods.

2. *Choice of premedication.*

Premedication rationally prescribed is based on two factors: (a) the patient's history and physical examination, and (b) anesthetic to be employed. One must therefore formulate the anesthesia before writing premedication orders. If the case is an emergency to be done immediately, the employment of intravenous premedica-

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tion should be seriously considered, and in my opinion is ordinarily most satisfactory, particularly if any element of shock is present or anticipated.

Premedication has four desirable ends: (1) to allay apprehension, (2) decrease pain, (3) suppress undesirable reflexes, suppress the production of secretions and reduce oxygen demands, and (4) counteract undesirable side effects of anesthetic agents. Ordinarily these are accomplished by appropriate dosages of: (a) a barbiturate to allay apprehension and decrease metabolism, (b) a narcotic to reduce pain and allay apprehension and further reduce metabolic oxygen requirements, and (c) a belladonna derivative to depress reflexes, and if scopolamine is used, to further allay apprehension and also to provide amnesia.

Which and how much of these drugs is to be used depends upon the age, metabolic rate known or estimated, physical state and allergic history of the patient, and as I have already noted, the anesthetic to be given. Regarding the anesthetic the following is generally true: if a general anesthetic is to be given one usually goes light on those drugs that depress respiration and heavy on those that depress reflexes. If spinal or regional are used, then cortical depression is desirable and reflex depression not so necessary. It should always be remembered that more of any of the premedicating drugs can be added intravenously if necessary to take care of emergency changes in plans or procedures but that an overdose can not be readily combatted. Particular care is necessary in the case of the very young or very old and in case of metabolic abnormality.

The child up to the age of about one year does not require heavy premedication. All that is desirable is a reduction of secretions and reflexes. This can be accomplished with atropine in doses of 1/800 gr. to 1/600 gr. Many anesthetists do not consider premedication of any sort required in this age group.

After one year psychic awareness becomes more acute and reactions of an indeterminable nature are more prone to occur due to changes in environment and because of unfamiliar personnel. Small doses of a rapid acting barbiturate given rectally tend to lessen apprehension and fright. Gr. ½ to 1 Seconal in the form of a suppository may be used 1½ hours before surgery. Atropine grs. 1/600 to 1/300 is given approximately 45 minutes before surgery. This type of premedication may be employed up to approximately the age of five or six.

In very agitated children, or where basal narcosis is required, pentothal in a 10 per cent solution administered in quantities of not more than 0.2 cc. per pound of body weight (4 cc. kg.) by rectum is very satisfactory. This is satisfactory in children up to 50 pounds (23 kg.) since not more than 1 Gm. of drug should be employed.

Avertin (tribromethanol in amylene hydrate) solution may be employed in the very young with considerable success. In these instances perhaps as much as 120 mg./kg. may be needed. This preparation may be given to patients of any weight or age. It is essential that the basal metabolic rate of the patient be known or estimated. Today this drug is used very little except for children, and since the B.M.R. of a child can only be estimated it is best that the Avertin solution be given fractionally, say one-half the calculated dose, then after ten minutes more is added, if required, so that no more is given than is necessary for the desired result.

Both rectal pentothal and Avertin are useful for doing many studies on children not involving surgery as such, X-ray studies, cardiac catheterization, encephalograms, pneumoencephalograms, etc. An enema should always precede the drugs and atropine in approximate dosages also employed.

In children over five years of age I employ demerol in doses of 25 to 50 mg., depending on age and condition, instead of a rectal barbiturate. In addition, scopolamine grs. 1/300 to 1/200 is given instead of atropine. This combination of demerol and scopolamine is very satisfactory in providing a quiet and cooperative child. Just when a child becomes an adult from a premedication point of view is difficult to determine. From about 10 to 15 years of age the metabolic rate is high and a great deal more premedication may be required than might be desirable from an age consideration alone. Seconal or pentobarbital may be employed orally in doses of gr. ¾ to gr. 1½ in older children and should be given 1 to 1½ hours before surgery.

From about 15 years of age to approximately 65—always remembering metabolic rate and physical condition the triad of a barbiturate—a narcotic and a belladonna derivative are used in appropriate doses. Seconal grs. 1½ to 3 or pentobarbital in the same quantities 1½ hours

before surgery with demerol 100 to 150 mg. or morphine gr. 1/6-1/4 and scopolamine gr. 1/100 to 1/150 one hour before surgery are most commonly used. There is one notable exception, and that is in the care of a patient undergoing a Cesarean section. These patients are unpremedicated except for a belladonna derivative if inhalation anesthesia is to be employed. Such patients are given any indicated sedation intravenously after the baby is delivered. It is amazing how many women will undergo the complete surgery of a Cesarean under spinal anesthesia totally unpremedicated and unsedated with the proper encouragement and assurances of the anesthetist.

When a patient reaches the age of approximately 65, the dosage of premedicating drugs should be reduced. The metabolic rates of these patients are low, their vascular respiratory reserve is reduced as a general rule, and frequently even small doses of sedatives and narcotics produce an undesirable depression. In these patients barbiturate dosage should be reduced to seconal gr. ¾ (or equivalent dosage of other preparations). Demerol is less depressing than is morphine and is substituted for morphine for all patients in this age group who are not suffering from acute traumatic pain.

Atropine is the belladonna derivative used for these patients since in this age group, as with the very young, scopolamine causes excessive restlessness in many instances. Demerol is 25 to 75 mg. doses and atropine gr. 1/200 are most often employed.

In emergency cases premedication can be given intravenously; in fact, because of the certainty of the absorption of the dose and the predictability of the time of the effects, many anesthesiologists prefer to give all premedication by this route. Morphine and demerol have their maximal depressing effects in less than ten minutes after intravenous administration. Analgesia is about maximal in fifteen minutes. Pentobarbital may also be given intravenously to the desired degree of narcosis very satisfactorily. The amounts of drugs given by this route are reduced over that given intramuscularly except for the belladonna fraction whose dosage remains the same.

The practice of "stealing thyroids" with basal narcosis with Avertin has almost disappeared and in fact the use of Avertin for any purpose for adults is very rare. The reason for this is the unpredictability of the degree of both respiratory and circulatory depression and duration of narcosis with Avertin. The more satisfactory intravenous sedation with pentothal or surital (thioanalog of seconal) has almost entirely replaced Avertin for basal narcosis in the adult when such is required.

There are other drugs that may be used for premedication or basal sedation in special instances.

Intravenous alcohol in 5 or 10 per cent concentration in glucose may be used to stabilize alcoholics. Paralydehyde or choral hydrate may be used in cases of acute mania or other allergic reaction to the usual premedication drug.

The premedication of brain surgery cases presents no serious problem today. Demerol, apparently, does not tend to cause an increase of intracranial pressure in therapeutically effective doses as morphine is said to do.

Severe toxic thyroid cases must be heavily premedicated frequently but can be brought to surgery with pentothal intravenously.

In cases where shock is present it is almost mandatory to give premedication intravenously to assure effect, since there may be a depot of drug in the tissues due to failing circulation. Later a severe depression may result as the circulation improves and the depot of drug is released into the circulation.

3. *Choice of anesthesia.*

The most commonly employed anesthesia for infants and children is open drop ether, or ether insufflation with air as the vehicle. Ether as an agent is well tolerated but the method of administration can be improved by supplementing the ether-air mixture with oxygen in the case of the open drop technique or by substituting oxygen for air in the insufflation techniques. An ether-air anesthetic always produces a reduction in the inspired oxygen and consequently in a reduction of the alveolar oxygen tension by a fraction proportioned to the concentration of ether vapor according to Dalton's law. While it is true ether causes stimulation of respiration, it is also very probable that part of the stimulation in the case of ether-air anesthesia is the result of hypoxia.

Ether anesthesia for children is usually induced with Divinyl ether or ethyl chloride because of their rapid action. Oxygen should also be used with these drugs because there is no doubt that the convulsions occasionally seen with Divinyl ether and the cardiac arrests re-

ported from ethyl chloride are hypoxic in origin.

Rectal pentothal plus nitrous oxide-oxygen—the oxygen percentage kept above 20 volume per cent—can be employed for infants and children with considerable satisfaction in many instances, particularly in orthopedic surgery, neurosurgery, and plastic surgery. Rectal pentothal plus topical anesthesia can be used for eye surgery with success.

Cyclopropane is mandatory for the induction of cases with Tetrology of Fallot for the Blalock operation since it allows a very rapid induction with high oxygen concentration of the inspired gases.

When properly premedicated, the use of spinal and regional anesthesia for surgery on children is entirely practical. Great care must be exercised not to use toxic quantities of drug or excessive concentrations. One-half per cent procaine solutions for regional is adequate. One mg. of procaine for every 1 kg. of body weight is adequate for spinal, and the spinal drug should be diluted to not more than a 2 per cent solution with spinal fluid before injection.

Because of the relatively high metabolic rate of children no hypoxia should be permitted. Endotracheal anesthesia may be safely employed to insure the airway provided care is taken to prevent laryngeal edema from too large a tube or from trauma as a result of rough technique. If a circle anesthetic gas machine is employed it is desirable to assist inspiration slightly by gentle rhythmic compression of the breathing bag to reduce respiratory effort of the small patient to a minimum, since labored respiration for any period of time may well result in collapse of respiration and circulation.

During prolonged anesthesia careful watch should be kept on the rectal temperature alterations in either direction from normal and be corrected.

The choice of anesthesia for adults depends upon many factors controlled by the patient, surgeon, or anesthetist. The patient is, of course, the most important control. Generally most patients will accept whatever anesthetic their surgeon and anesthetist feel is best. Under no circumstances should a patient be given an anesthetic he does not want, particularly a spinal anesthetic, unless it is absolutely necessary.

The majority of patients are perfectly able to take any of a variety of anesthetics, but as has been pointed out, it is essential that a physiological and physical assay be made before any patient is anesthetized by any agent and method.

The majority of surgeons will accept the anesthetic chosen by the anesthetist. The anesthetist should, however, always try to choose the type of anesthesia most suited to the surgeon if there is no reason why such can not be employed. In other words, the choice should be a cooperative effort. If for any reason there is a difference of opinion this can almost always be resolved by a review of the patient and his record by the surgeon and anesthetist together.

It is the opinion of most anesthesiologists that spinal anesthesia should not be used for the shocked patient or one who may go into shock. Spinal anesthesia is also best avoided in patients with concealed hemorrhage such as ruptured ectopic pregnancies.

Regional anesthesia is ideal for the patient in shock, if he must receive surgery. Cyclopropane is the anesthetic of second choice. Intravenous morphine or demerol and nitrous oxide-oxygen may be employed, using a minimum of pentothal. Deep ether anesthesia is to be avoided.

A word of caution regarding general anesthesia in the emergency case is in order. Try to ascertain how long before injury or the onset of the acute process the patient has had food. Invariably gastric function stops after injury or with severe acute pain. It is very important that the stomach be emptied if undigested food is reasonably suspected to be present. More tragedies have occurred from the vomiting of food by emergency patients during some phase of an inhalation anesthesic with aspiration into the lungs of an undetermined quantity of the material than from any other factor due to anesthesia. It may be unkind to subject the injured or acutely ill patient with a full stomach to gastric lavage or emesis, but in actual fact he will invariably vomit sooner or later and it is better that it be done under controlled conditions.

Theoretically, the anesthetist should be expert with all techniques. This is not always the case for a variety of reasons. The inexperienced anesthetist should not attempt new or unfamiliar methods without experienced guidance. This he owes to the patient.

The aged patient, as the very young patient, requires special consideration. The aged patient has lost a very large part of the respiratory and vascular adaptability that is possessed by the patient of less years. It is therefore extremely

important that respiratory and vascular depression be avoided. In this age group utilization of multiple agents, avoiding the undesirable characteristics and taking advantage of the desirable ones, is best employed. Regional anesthesia with light sedation can be used for many procedures. Nitrous oxide-oxygen and very small doses of demerol intravenously provide excellent anesthesia for many patients. Spinal anesthesia, if a unilateral type can be used, is very satisfactory, particularly for fractured hips. Deep anesthesia should be avoided where possible. Small doses of curare can be employed to secure adequate relaxation with a minimum of undesirable depression, with very light general anesthesia.

Certain principles, then, should be observed in the choice of anesthesia, namely:

(a) There is no such thing as choosing anesthesia for a particular type of surgery—anesthesia is chosen for the patient only. It is true that most patients can take any number of a variety of anesthetics without any noticeable difference, and in these the most convenient anesthetic for the surgeon should be employed, but in certain cases no compromise should be made.

(b) No patient should be given an anesthetic he does not want or is afraid of. This is particularly true of spinal anesthesia. Most patients want to be asleep during surgery and frequently a spinal will be accepted if the patient is assured he will be asleep.

(c) If a patient states in all seriousness he will never wake up if he is given a general anesthetic and that he is going to die on the table, cancel the surgery or at least decline the responsibility of giving the anesthetic, because the patient is right in about 95 per cent of the cases.

(d) The facts must be faced that all anesthetics are potent protoplasmic poisons. They constitute some of the most potent drugs in the pharmacopia and it is indeed unwise to take lightly the responsibility for their administration.

Conclusion.

It is not good anesthesia merely to have a patient survive an anesthetic; fortunately most patients will survive no matter how poorly chosen or administered is the anesthesia. Failure to properly evaluate the importance of anesthesia in the past is why we are now struggling to revive this branch of medicine from the status of a technical maneuver to which it had degenerated. The function of an anesthesiologist is to so choose and administer an anesthetic that the patient is no worse off for having received it and, if possible, as is often the case, by fulfilling his corollary duties of providing adequate hydration, electrolytes, and blood replacement, that the patient is actually improved after surgery over his preoperative condition.

An anesthetic well given therefore starts with an adequate preoperative evaluation of the patient derived from personal contact with him, with a review of his history and a physical examination, is continued by intelligent preoperative medication, and terminated by finally administering the actual surgical anesthesia.

THE SELECTION OF ANESTHESIA FOR THE CARDIAC PATIENT*

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The selection of anesthesia for patients with cardiac disease has always presented a problem. Many of these patients are geriatric subjects to whom a major operative procedure plus the trauma of anesthesia may lead to an unfavorable outcome[1]. We do not delude ourselves that anesthesia is not trauma.

In the modern concept of anesthesia it is not considered enough merely to keep the patient alive on the operating table and to get him back to his room. The anesthetic should be so chosen and conducted as to give the patient the best possible chance of recovery. The anesthetic agent and technique should be the one most physiologically sound for the individual patient. This choice is arrived at by taking into consideration the pharmacology of the agents used and the physiological state of the patient at the time of operation. Deaths which occur in the first post-operative days may be due to the way in which the anesthesia was conducted. The patient may be so weakened by the bad effects of the anesthesia that his downhill course is accentuated and ends in his death[1].

In minor surgery where little or no relaxation is required the choice of anesthesia is local infiltration or a regional block. This produces the least metabolic effect upon the patient. When this is not practical, he may be given small doses of pentothal as a basal anesthesia supplemented with nitrous oxide, and oxygen in large concentration to provide analgesia. He may receive a regional block for analgesia and pentothal in small doses to increase his comfort. Oxygen should always be given. This type of anesthesia is hardly sufficient for some types of intra-abdominal surgery. There remains for these cases inhalation or spinal anesthesia.

The types of heart diseases most commonly encountered are coronary artery insufficiency, cardiac decompensation and hypertension.

CORONARY INSUFFICIENCY

The patient with coronary artery disease is usually apprehensive about his heart, since he has had painful attacks. These people must receive adequate premedication to allay their fears but they must not be over sedated. The doses of medicine must be individualized after separate evaluation of each case. Routine orders should never be given for these patients. If the patient is excessively nervous he may be put to sleep with some pentothal before he enters the operating room.

Four chief dangers are to be avoided in patients with real or potential coronary insufficiency

(1) Anoxia of the cardiac muscle must be prevented during a fall of the diastolic blood pressure since the diastolic pressure level determines the coronary artery blood flow.

(2) A high oxygen concentration must be maintained.

(3) The muscular exertion of the excitement stage must be avoided because of the possibility of coronary spasm with occlusion and myocardial infarction. This may be overcome by the use of rapid acting agents such as pentothal for induction.

(4) Avoid an overload to the circulation due to the administration of large amounts of fluids[1].

Spinal anesthesia is contraindicated in most cardiac cases because of the tendency to produce a drop in pressure. Vasopressor drugs and oxygen may bring the pressure back but a severe drop of the blood pressure may produce disastrous results before it is remedied.

For intra-abdominal surgery there are several method of obtaining relaxation while fulling the four conditions just mentioned.

(1) One excellent method of doing this is with an intercostal block done while the patient is asleep with pentothal oxygen, or pentothal–nitrous-oxide-oxygen. If the surgery is in the lower abdomen a field block may be done or the muscle infiltrated with one percent procain. This may completely eliminate the necessity for the use of curare or ether.

(2) The blocks or infiltrations may also be used with pentothal induction and cyclopropane. This produces relaxation and eliminates the need for deeper anesthesia.

(3) Ether in small quantities added to cyclopropane after pentothal induction is very useful. It provides high oxygen concentration, a pleasant quick induction, and the ether stabilizes the

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heart rhythm and provides relaxation. The patient will be unaware that he has had any ether because the quantities used are small.

For any of these combinations a small amount of curare may be used. I should like to add here a word of caution about the use of curare. When adequate relaxation can be obtained by other means such as infiltration, block, or the addition of small amounts of ether, the use of curare should be very limited. It is important to maintain the mechanics of the respiration to produce the proper filling of the right heart. The proper filling of the heart is necessary to maintain the cardiac output, coronary circulation and blood pressure. During inspiration, by the elevation of the ribs and descent of the diaphragm a negative pressure is created inside the chest, which is transmitted to the right heart. This negative pressure plays a very important part in cardiac filling, therefore on the cardiac output and the maintenance of blood pressure. Except when dealing with a very flabby individual, enough curare to produce abdominal relaxation will paralyze the intercostal muscles. At least half of the negative pressure produced by the inspiratory movements will be lost. The chest may be inflated by pressure on the breathing bag of the gas machine to provide adequate ventilation but the positive pressure required to inflate the chest is also transmitted to the right heart and prevents the adequate filling, to some degree. This degree is insignificant in the normal or near normal individual but may have a definite weakening effect upon the patient with coronary artery insufficiency or hypertension.

CARDIAC DECOMPENSATION

In dealing with cardiac decompensation we are concerned chiefly with pulmonary edema. The etiology is controversial. The problem of the anesthesiologist is to alleviate the symptoms of pulmonary edema or avoid aggravation of those already present[1]. In this group are the younger patients of the rheumatic heart disease group. These patients have a history of cardiac decompensation or signs and symptoms of its presence. Those who show findings which suggest that an overload to the circulatory system might precipitate decompensation must be included in this category. Dyspnea on exertion, ankle edema and orthopnea are the most prominent symptoms and basal rales may be found on ausculation[1].

The choice of anesthesia in this group of patients is spinal. This conclusion is based upon the work of Sarnoff and Farr[2] in using spinal anesthesia for the treatment of pulmonary edema in cardiac decompensation.

These investigators chose patients admitted to the hospital in severe cardiac failure with pulmonary edema as the predominant symptom causing distress. Spinal anesthesia was given to cause peripheral dilatation of blood vessels and muscular relaxation in the caudad portion of the body. Some of the blood is trapped there, reducing the circulating blood volume and load on the heart. The anesthesia acts as a bloodless phlebotomy in the same manner as applying tourniquets to the extremities. There is a reduction in the venous return to the heart which aids in correction of the imbalance between the right and left heart. This facilitates return of fluid in the lungs to the general circulation and relieves the pulmonary congestion. Other accepted medical treatment was carried out at the same time[1-2].

When surgery is to be done below the diaphragm spinal anesthesia is a good choice. If the operation is to be a prolonged one the fractional technique may be carried out.

In the patient who presents pulmonary edema, general anesthesia of sufficient depth to provide relaxation may precipitate severe cardiac failure with marked increase in the pulmonary edema. In some cases though the patient may go through surgery and the operative day with no increase in symptoms or physical findings, the heart failure may begin to manifest itself and progress during the first and second post operative days. Anesthesia can still be considered a contributing factor here[1].

If the operation is elective and the patient presents some signs of heart failure he should be digitalized before surgery is attempted. In the opinion of some internists, strictly elective surgery should not be carried out on patients who have a history of cardiac failure. The hazard is great and should be done only as an emergency arises or to prevent an emergency.

As soon as a spinal anesthesia has been given oxygen should be administered in high concentration preferably thru the gas machine where respiratory movements may be closely observed in the breathing bag. Oxygen should be continued through the post operative period as long as heart failure or its imminence is present.

It is essential to use, routinely, a vasopressor drug to maintain the blood pressure. Since neo-

synephrin produces a bradycardia, does not cause arrhythmia, and increases the stroke volume it is a good choice for this purpose. Ephedrine is also acceptable. Pitressin and pituitrin are especially to be avoided since they are powerful coronary vasoconstrictors. Coronary vasoconstriction leads quickly to myocardial anoxia, cardiac dilation, fall in blood pressure and even sudden death[3-4].

An intravenous solution of dextrose and water should be started before the operative procedure but it should drip very slowly. This is mainly to assure a readily accessible vein for the administration of intravenous medication or blood should the occasion arise.

HYPERTENSION

Patients with hypertension are treated much the same as those with coronary artery insufficiency. The main difference in the treatment of the hypertensives is consideration of the elevation of the diastolic blood pressure. As previously stated it is upon the maintenance of the diastolic pressure that the coronary arteries depend for their blood supply. A severe and abrupt fall in diastolic blood pressure may so embarrass the heart through anoxia that it will dilate and cease to function.

Spinal anesthesia is definitely contraindicated for any intraabdominal procedure in the group with the high diastolic pressure. Where the anesthesia may be limited to the saddle areas it may safely be used, since this will produce a negligible drop in the blood pressure. If a saddle block is used it is wise to use 100% oxygen during the procedure[1] and it is also wise to consider a general anesthetic of some type.

Where relaxation is required for major procedures cycloprepane-oxygen with small amounts of ether will produce less systemic change than any of the other inhalation agents. If the patient has an arrhythmia or a history of an arrhythmia then another choice should be made. An induction with pentothal to prevent the excitement stage, followed by the careful addition of ether and oxygen through the gas machine is an excellent choice. By this combination, carbon dioxide absorption may be carried out and a much higher percentage of oxygen may be used than in the nitrous-pentothal-curare sequence. In my opinion ether-oxygen with pentothal induction is the anesthesia of choice for the patient with hypertension who has an arrhythmia where relaxation is very important. Ether must be very carefully added after a pentothal induction to prevent a laryngospasm. The patient must be lightly premedicated. This means no preoperative barbiturate, and if the patient is over 60 years of age no more than ⅛ grain of morphine and an appropriate dose of atropine or scopalamine. This combination should be given at least one hour before the induction of the anesthesia so that it may reach it maximal depressing effect upon the respiratory center before induction of the anesthesia. Many times the elderly patient will need no more than 1/12 grain of morphine or its equivalent in Demeral of 50 milligrams. If the patient's age is in the 70's or 80's codein may be substituted for morphine. These patients should be awake and moving as soon as possible after the surgery and if they have had barbiturates, and a dose of morphine which is large considering their metabolic state, they are far too depressed.

It is possible to use pentothal, nitrous-oxide-oxygen-curare but caution must be taken not to depress the respirations with the curare. It is better to use a block or infiltration with procain and then a very little curare if more relaxation is needed.

The only time open drop ether is indicated is in an emergency when a trained anesthetist is not available. If ether must be given by an inexperienced person under the direction of the surgeon, oxygen should also be given by nasal catheter or by direct flow under the ether mask and it should be started with the induction.

In summary—we must remember that cardiac cases during anesthesia (1) do not tolerate blood pressure drops and everything must be done to maintain the blood pressure. (2) A high oxygen concentration must be continued. (3) They must not be given drugs which will render the myocardium anoxic. (4) In so far as is possible the normal mechanics of respiration must be maintained.

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THE DIFFERENTIAL DIAGNOSIS OF HEMORRHAGE FROM THE UPPER GASTRO-INTESTINAL TRACT

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One of the most common sites of symptomatic bleeding is the gastrointestinal tract. Hemorrhage from this source is manifest by hematemesis, the vomiting or regurgitation of blood, or by melena, which is the discharge of black modified blood from the bowel.

The exact frequency of hemorrhage from the gastrointestinal tract can, of course, only be surmised. Many persons will pass black stools and some few will actually vomit appreciable amounts of blood without seeking medical opinion. Some worthwhile statistics on the frequency and etiology of gastrointestinal hemorrhage have been reported, however, from larger teaching hospitals where such cases are more likely to be concentrated.

Thompson, et al.,[1-2] recently reported on 557 patients admitted to the Los Angeles General Hospital in one calendar year for bleeding from the gastrointestinal tract. The number of cases is not sufficiently large that each possible cause of hemorrhage is represented, but it does give some idea of the magnitude of the problem. This study was reported in two separate papers, one on hematemesis, and the other on melena.

Wilbur and Rivers[3] reviewed the records of 668 patients admitted to the Mayo Clinic with a history of vomiting blood. It was found that in 56.6 per cent of the cases the hematemesis was caused by duodenal ulcer. Welch[4] recently reported on the source of bleeding in 363 cases of massive acute hemorrhage from the upper part of the gastrointestinal tract observed at Massachusetts General Hospital from 1938 to 1947.

Any attempt to classify gastrointestinal bleeding on the basis of the site of the lesion must be founded on a knowledge of anatomic and pathologic abnormalities encountered at various levels of the alimentary canal. This can be divided roughly into three segments: from the pharynx to the ligament of Treitz, from the ligament of Treitz to the ileocecal valve, and from the ileocecal valve to the anus.[5] In this discussion we are interested only in bleeding from the first two divisions.

The lesion producing hematemesis generally is situated proximal to the ligament of Treitz, while the lesion producing melena probably is located above the midpoint of the small intestine. Bleeding below this point is usually evidenced by red blood in the stools.

There are exceptions to these rules, however:

1. Even with esophageal bleeding the blood may not be vomited and hemorrhage may be suspected only because of tarry stools.

2. Although melena is the rule in upper gastrointestinal tract bleeding, it must not be forgotten that feces containing currant jelly-like, deep reddish material may be encountered if the hemorrhage is brisk and the peristaltic rate rapid.

3. Bleeding distal to the ligament of Treitz, although usually manifest by melena, may, in the presence of intestinal obstruction with reversal of the intestinal peristaltic gradient, produce hematemesis.

The typical tarry stool is viscid, shiny, and black. Clinically it must be differentiated from the black discoloration of feces which follows the oral administration of iron, bismuth, or charcoal, or from the dull red stool which sometimes appears after the ingestion of highly colored foods. Chemical tests are of assistance in making the distinction. Daniel and Egan[6] have demonstrated experimentally that as little as 50cc. of blood placed in the stomach may cause a tarry stool.

Consider now the causes of upper gastrointestinal hemorrhage. Esophageal bleeding is due either to varices or to cancer in most cases. In the Los Angeles County Hospital study varices accounted for 32.2 per cent of the cases of hematemesis and 9.2 per cent of the cases of melena. The presence of esophageal varices secondary to portal hypertension may be suspected from the observation of jaundice, spider angiomata, ascites, or the finding of a palpable liver or spleen. A history of chronic alcoholism in some of these patients may be significant. Carcinoma, the commonest malignant tumor of the esophagus, usually manifests itself by dysphagia, epigastric pain, anorexia, weight loss, and inanition. If

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ulceration is present, however, hemorrhage may occur.

Peptic ulcer is the commonest cause of massive hemorrhage from the upper gastrointestinal tract. According to Bockus[7] approximately 75 per cent of all massive hemorrhage into the alimentary tract is caused by peptic ulcer. The great majority are located in the first portion of the duodenum.

The history of active ulcer symptoms preceding the hemorrhage with the sudden cessation of all pain after the onset of bleeding justifies the tentative diagnosis of peptic ulcer.[8] A previous diagnosis of peptic ulcer or the history of chronic intermittent ulcer distress over a period of years with recurrence of discomfort shortly before the onset of hemorrhage indicates a peptic ulcer as the probable source of hemorrhage.

Objective evidence of peptic ulcer obtained by previous roentgenologic examination is valuable, not only in establishing the diagnosis, but in localizing the lesion to the exact site in the stomach or duodenum.

Approximately 25 per cent of patients, however, deny the existence of ulcer symptoms prior to the hemorrhage. This is particularly true in the older age group. Not uncommonly the patient presents no history or roentgenologic evidence of a previous peptic ulcer, and studies performed in one-fourth of the cases of massive hemorrhage within three weeks of the onset of the hemorrhage fail to establish the cause and site of the bleeding.

Cancer of the stomach is not a common cause of massive gastric hemorrhage and usually occurs in the late stages of an ulcerating carcinoma. Evidence of weight loss and the finding of an abdominal mass suggest the diagnosis. Polyps of the stomach and other benign tumors may cause massive hemorrhage without warning. The diagnosis may be established by roentgenologic or gastroscopic examination.

Severe hemorrhage from the stomach may originate from a hypertrophic gastritis, an erosive hemorrhagic gastritis, or superficial gastric ulcerations. Fairlie has written a recent article in which he reviews four cases of gross hemorrhage due to chronic gastritis. These were of the hypertrophic and superficial types. Gross hemorrhage as a result of atrophic gastritis is described but is certainly far less common than that from the hypertrophic and superficial types.

Hematemesis or melena in an elderly stout woman suggests a hiatus hernia, particularly in the absence of typical ulcer symptoms. The hemorrhage is initiated in these instances by gastritic erosions, ulcerations, or severe hypertrophic gastritis within the herniated pouch.

Hodes and Edeiken[9] recently published a comprehensive study on roentgen manifestations of small intestinal bleeding. Of the 161 patients in this study there were seven patients with non-Meckelinian diverticulae. These are found most often in the second portion of the duodenum, less commonly in the jejunum, and least commonly in the ileum. Usually found penetrating the muscular layers of the small intestine at points where blood vessels enter, it often contains ectopic pancreatic tissue. It is host to all the complications found in Meckel's diverticulum and bleeds with equal severity. Benign and malignant tumors, tuberculous and non-tuberculous enteritis, and jejunal and ileal ulcers are less common causes of intestinal bleeding.

Of the latter small bowel lesions let me emphasize only three: regional enteritis, nonsclerosing ileitis, and, of the malignant tumors, lymphosarcoma.

As Hodes and Edeiken pointed out regional enteritis is characterized by a non-specific sclerosing inflammatory process that involves the terminal ileum, ileocecal region, or any portion of the small intestine. It often is associated with a narrow lumen, hypertrophy of the muscularis propria, mucosal erosions or ulcerations, and fistulae. The radiological findings are characteristic.

The nonsclerosing affection, or ileitis, occurs more commonly in girls early in life. Pain in the lower right portion of the abdomen, often mistaken for appendicitis, is the outstanding symptom. Radiological findings are right lower quadrant tenderness and an irregularity of the mucosa of the terminal ileum sometimes referred to as a cobblestone ileum.

The location of preference for lymphosarcoma is the terminal ileum possibly because of its abundant lymphoid structure. Yet these lesions are not uncommon higher in the small bowel involving the duodenum and proximal jejunum. The result of the disease on the bowel is peritonitis, adhesions, obstructions, necrosis, and hemorrhage.

Meckel's diverticulum is responsible for many serious and unrecognized abdominal complications. As a rule it is located on the antimesenteric

border of the ileum 30 to 90 cm. above the ileocecal valve. It is twice as common in males as in females and occurs in about 2 per cent of the population. Hemorrhage is one of the most common symptoms of diverticulum. It occurs more often in persons under 15 years of age and especially in infants under 2 years. The bleeding is attributable to the ulcer, erosion, and inflammation that develops in the heterotopic tissue within the diverticulum. Usually the ulcer may be found at the junction of the heterotopic mucosa and the normal mucosa.

Blood dyscrasias should be suspected whenever multiple sources of hemorrhage are observed or splenomegaly of undetermined origin is found. Thrombocytopenic and non-thrombocytopenic purpura, splenic anemia, and hemophilia are possibilities.

Among the intrinsic causes of bleeding from the small bowel are vascular diseases, such as thrombosis of mesenteric arteries and veins, rupture of arteriosclerotic blood vessels, or peptic erosions secondary to endarteritis.

Finally foreign bodies, allergies, and drug idiosyncrasies may be causes for a rare and obscure case of upper gastrointestinal hemorrhage.

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Editorials

PRESENT STATUS IN THE TREATMENT OF TUBERCULOSIS OF BONES AND JOINTS

W. A. BISHOP, JR., M.D.
Phoenix, Arizona

Tuberculosis of the bones and joints is one of the oldest phenomena of which there is record in the medical archives. Through the ages, there has been a slow evolution in the treatment of this condition. Originally, the major medical centers over the world considered that tuberculosis of bones and joints should be treated conservatively. Immobilization over a long period of time with eventual ankylosis of the joint or healing during the wearing of a brace was accepted as the treatment of choice until the era of asepsis.

Soon after it became possible to operate upon bones and joints—particularly diseased bones and joints—without great danger of infection, there were enthusiasts for early operation to secure an arthrodesis in all cases of tuberculosis.

During the past twenty-five or thirty years, there has been a tendency for the pendulum to swing to the more conservative and then to the more radical form of treatment. It has been the consensus of most orthopedic surgeons during the past ten or fifteen years, that the judicious use of conservative treatment until the condition was quiescent, prior to surgical intervention, would give the best results in the greatest number of patients.

These patients were treated much as any patient with tuberculosis, by rest and immobilization of the part until the patient's general condition improved and until the reaction in the joint was quiescent. A surgical arthrodesis was then attempted with the ultimate goal of a bony ankylosis of the joint to eliminate motion and

consequently to permit relatively normal activity without the great danger of repeated flare-ups.

More recently, Streptomycin has been used over a period of time and produces dramatic results as compared with the older conservative methods, particularly when applied to conditions with draining sinuses and secondary infection. By a combination of Streptomycin and whatever other antibiotic that would be indicated for the contaminating organism present, most of these conditions clear relatively rapidly, and permit surgical intervention at a much earlier stage than has been possible heretofor.

It is too early in the history of Streptomycin, and some of the other drugs of promise, to be able to say if the condition of tuberculosis of bones and joints will lend itself to chemotherapy without other measures. At this writing, it would appear that chemotherapy is merely an adjunct in the treatment of the disease.

Through evolution in the treatment of chest conditions, there has gradually evolved a method by which patients with localized, far advanced disease are subjected to surgery in the form of lobectomy or other measures for eradication and elimination of the disease process. All too frequently, we see patients with chronic draining sinuses of many years duration following unsuccessful treatment of tuberculous infections of the bones and joints. One should not lose sight of the fact that eradication of the focus is often indicated in these conditions, both as a measure of permitting physical rehabilitation, but also as a means of preventing amyloidosis. An amputation should always be considered in chronic tuberculous bone and joint conditions which have not responded to a reasonable period of less radical treatment.

In conclusion, it may be stated that the treatment of tuberculosis of the bones and joints has made gradual progress through the decades with swinging from ultra conservative prior to the period of asepsis in surgery, to marked enthusiasm for early surgical treatment. This was followed by a swinging back to a more conservative approach, with surgical intervention only after the patient's general condition, and the local infection had shown improvement. Since Streptomycin and the other promising drugs have been available, many patients with draining sinuses have been cleared up, and surgical intervention has been possible in patients in which all previously known therapeutic measures had failed.

Extirpation of the lesion by amputation is necessary in a certain number of patients to eradicate the disease process from the patient when that procedure alone offers hopes for rehabilitation of the more chronic conditions.

ARIZONA MEDICAL PROBLEMS

Consultation and Case-Analysis

ARIZONA MEDICINE again presents an unsolved and difficult case from the practice of Arizona physicians, with the Case-Analysis and comments of a specially-chosen and nationally-known Consultant.

Any physician who has an undiagnosed case which has defied other methods of solution may send it for consideration. The case should be completely worked up, but an editor will help compose the report. Whenever the need for an answer is urgent, the Consultant's reply will be sent direct to the submitting physician, before publication.

Please send communications and data to Dr. W. H. Oatway, Jr., 123 S. Stone Avenue, Tucson, Arizona, or care of The Editor, Arizona Medicine.

The *current case* represents a common accident in Arizona. It is a probable medical and surgical case with orthopoedic and thoracic aspects.

The *consultant* is Dr. Hobart A. Reimann, Professor of Medicine at The Jefferson Medical College of Philadelphia.

Dr. Reimann is ideally qualified to handle this case-analysis. His interests are very diverse, and he has published interesting papers on widely separated subjects.

As a possible connection to the case at hand, he is the author of a book on pneumonia, of several sections in *Cecil's 'Textbook of Medicine,'* including Circulatory Disturbances of the Lung, Pulmonary Edema, Pulmonary Hemorrhages, Pulmonary Concussion, Pulmonary Infarction, Atelectasis, Abscess, etc., and author of a four-volume set on treatment in internal medicine.

CASE NUMBER XXV

(*The following history is restricted because of language difficulties, the short duration of the illness, and the patient's serious condition.*)

The patient was *a white male Spanish-American* who appeared younger than his 72 *years of age.* He was a ranch-foreman, and led a very active life. He was said to have been well until his recent accident.

The patient was thrown by a horse, and rolled-on. He was carried to his home on the edge of town, put to bed, and a surgeon was called.

Examination showed *a crushing of the right side of the chest,* abrasions of the skin, but no other gross injuries. Because of shock and pain he was kept in bed at home *with his chest widely strapped* with adhesive-tape. He refused hospitalization.

X-rays of the chest were made at the bedside after 4 days, and they showed that six of the ribs on the right (3rd to 9th) had one or more fractures laterally. Other details were not clear.

On the sixth day after the injury the patient's cough and pain were increased, and he began to have *blood-streaked sputum,* a more severe dyspnea, grunting respirations, and a pain in the *left* lateral chest area.

The following evening (at the end of a week) the pain on the left side became more severe, and *he was seen in consultation.* He was quite cyanotic, in considerable respiratory distress, but the streaking of the sputum had ceased. His temperature was 100.2 deg. (oral), his pulse was 120/min. with occasional skipped beats, and the respiratory rate was 30/min. Rhonchi could be heard (thru the adhesive-tape) in each lung; there were possible signs of a pneumothorax on the right, with distant breath sounds; and the mediastinum seemed shifted to the left. His blood pressure was 100/70. The heart sounds could be heard only indistinctly. The abdomen and legs were normal.

He was given morphine. It was suggested that the tape be removed. A family council was to be held to urge hospitalization.

Early the following morning he had a sudden *sweat,* had the appearance of *shock,* and became *stuporous.* He was then taken to the hospital.

At the hospital he continued to be stuporous; his breathing was noisy, labored, and of a Cheyne-Stokes type; he occasionally coughed, choked, and raised profuse bright red sputum; and he perspired profusely, with a cold face and warm extremities. The pulse was 120-134, respirations 18-20, and temperature dropped from 100.8 to 99 deg. during the first day.

There was a subcutaneous emphysema of the right lateral chest, and the area was grossly discolored and swollen. There were moist coarse and medium rales throughout both lung fields, though they were distant on the right.

An X-ray at the bedside showed nothing unusual about the heart or mediastinum. There was *emphysema of the right chest wall, but no pneumothorax.* The multiple, complete, ununited fractures were as previously noted. *There was a generalized soft patchy accentuation of the trunks in both lung fields* which suggested a 'pulmonary edema or hypostatic pneumonia' to the radiologist. There was also a slightly greater density at the left base, suggesting pleural effusion.

A blood count included 12 grams of hemoglobin, 3,680,000 R.B.C., 15,400 W.B.C., 90% P.M.N., and 10% lymphocytes. The urine was noted to be very concentrated, with 1.034 sp.gr., a trace of albumen, a few W.B.C. and R.B.C. per H.P.F.

Therapy consisted of oxygen by nasal catheter, coramine I. M., cedilanid I. V., and intravenous hypertonic glucose. Penicillin was not started until the temperature began to rise.

The patient roused somewhat during the next 12 hours, but he remained weak and restless. His pulse was thready and slightly irregular at 120 to 130/min. Expectoration of clotted blood and purulent sputum continued, and the respirations became regular but shallow. The *temperature* began a regular progressive rise which eventually reached 107 deg. The blood pressure dropped from 104/66 to 90/70 to 60/40. In spite of the continued regulated use of coramine, cedilanid, and finally adrenalin, he expired quietly 36 hours after entering the hospital. *A necropsy was not allowed.*

The diagnosis was not clear, and an analysis has been requested. It would seem that he either had a condition which would not respond, or else that the right things were not done.

QUESTIONS:

1. (a) What is the probable cause of the lung changes? (b) What is the cause of the cardiac failure?
2. What complications are liable to follow such an injury?
3. What could have been done in a diagnostic way? For therapy?

M. D., Tucson

CONSULTATION AND ANALYSIS
BY DR. REIMANN—

A man, aged 72, received severe trauma to his chest. Examinations showed "crushing of the right side," *but to what extent?* Was the thorax actually deformed with depression of the fractured ribs, or was there only superficial contusion? If ribs were broken and depressed one might have suspected *immediate laceration* of the pleura and/or lung with hemothorax and/or pneumothorax, and/or hermorrhage of the parenchyma of the lung. Since roentgenography revealed fractures only, and for 6 days no blood was expectorated, it must be assumed that the conditions mentioned did not occur. If the man were "thrown by a horse" and fell heavily to the ground striking the right side of his chest, *concussion of this right lung with diffuse interstitial or alveolar hemorrhage* is a more probable possibility. *Rupture of this lung occurred,* at any rate, to account for emphysema of the skin and muscles noted clinically and roentgenographically. That some *bleeding* occurred is suggested by the slight degree of secondary anemia later.

Contusion pneumonia of the right lung must be considered, but usually several hours and days elapse before characteristics of pneumonia develop. However, because of the patient's age and injury, pneumonia, if present, may have been indolent. Cough, dyspnea, slight fever and the rales which were mentioned are compatible with any of the conditions mentioned. Roentgenography did not give evidence of pneumothorax, pleural effusion or involvment of the right lung up to this time.

On the sixth day cough and dyspnea increased and bloody sputum was raised. In addition, pain developed in the left side of the chest. Next day the symptoms were intensified; no blood was raised but fever then was reported. There were rhonchi in both lungs and a possible shift of the mediastinum to the left. *The following possibilities may be mentioned to account for the increased degree of illness*:

The left lung may have been involved from (a) *pneumonia by contrecoup with pleuritis;* (b) *aspiration* of blood and secretion from the right lung, causing aspiration pneumonia; (c) *hypostatic pneumonia* from injury and confinement of an old person in bed; (d) *atelectasis* from immobilization in bed or from tight strapping; (e) *infarction* from immobilization or from emboli shed elsewhere; and (f) *congestion and hypostatic* pneumonia from circulatory failure or heart failure.

Fever, cough, dyspnea, cyanosis, bloody sputum, and leukocytosis are common to any of the 6 possible conditions listed. While *pneumonia* most likely was present, it probably was *secondary* to the other circumstances. *Atelectasis,* suggested by clinical evidence of shift of the mediastinum to the left (in the absence of right sided pneumothorax) also could have been secondary to any of the factors mentioned in the preceding paragraph.

The most likely cause of the condition, however, seems to have been *either circulatory failure or heart failure.* With the evidence at hand it is not possible to distinguish between the two, except that the *absence of peripheral edema suggest primary circulation failure.* This circumstance is not uncommon after severe injury in old persons. In this case failure may have been the result either of senescence or cor pulmonale from increased resistance in the pulmonary circulation. A combination of circumstances most probably was responsible. Evidence of failure began in the sixth day with tachycardia, irregular pulse, indistinct heart sounds, rhonchi in both lungs, low blood pressure (assuming it was higher before the accident) and concentrated urine.

The *sudden collapse and shock-like state* which took place in the eighth day might have resulted from several causes, as follows: (a) *embolism* of the lung, (b) sudden *atelectasis,* or, most likely, (c) final *collapse of a failing circulatory system.* The large amount of bright red sputum could have been caused either by infarction or circulatory failure. Coughing, sweating, tachycardia, fall of temperature and blood pressure, and a normal respiration are characteristics of *circulatory failure.* A roentgenologist reported possible edema of lungs or hypostatic pneumonia because of accentuation of the trunks in both lung fields, but these shadows also are suggestive of *venous overfilling* from circulatory failure. Density reported as possible effusion in the left base could have been due to atelactasis or hypostatic pneumonia.

The reported leukocytosis was compatible with any of the conditions mentioned but probably indicates secondary invasion of the congested, injured lungs.

QUESTION 2: *What could have been done in a diagnotic way?*

ANSWER: *More clinical information* about the first 6 days would have been helpful. If fluid was suspected in the left pleural cavity, *aspiration* might have been revealing. *Hydrothorax* from circulatory failure, *hemothorax* from an infarct, or *empyema* secondary to pneumonia might have caused it. *Bacteriologic examination* of the sputum would have been an aid to diagnose pneumonia if a predominance of pneumococci or other pyogenic bacteria had been present. Measurement of the *venous pressure*, of the corpuscular volume, and an electrocardiagram may have revealed the status of the circulatory system.

QUESTION 3: *What could have been done therapeutically?*

ANSWER: The questions of immobilization in bed, and of tight "strapping" of the chest is a problem in old patients. Both are conducive to *thrombosis and debilitation.* Yet after severe trauma there is often no choice. Tight strapping of the thorax leads to hypoventilation, anoxemia, cyanosis, embarrassment of the circulation, congestion of the lungs, atelectasis and local infarction; especially in debilitated patients. Yet, again, in the present case, severe pain may have necessitated a degree of immobilization. Morphine, often necessary to control pain, may be contraindicated in old people. Penicillin was indicated empirically because of the suspicion of pneumonia.

Treatment of circulatory collapse is indicated in this case; generally is unsatisfactory. It would be better to be able to control the condition which led to it. The injection of fluid sometimes causes overdistention of an already embarrassed circulation. The problem, unless dehydration is present, is not so much one of adding fluid, as it is to *get the blood to circulate properly.* The commonly used so-called cardiac stimulants, such as coramine, camphor, atropine, or strychnine rarely are helpful, and may cause harm. Cedilanid and digitalis are indicated only if actual heart failure or auricular fibrillation are present.

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PHOENIX CLINICAL CLUB

The Case History in this discussion is selected from the Case Records of the Massachusetts General Hospital, and reprinted from the New England Journal of Medicine. The discussant under Differential Diagnosis is a member of the staff of the Massachusetts General Hospital. The other discussants are members of the Phoenix Clinical Club.

M.G.H. CASE

A thirty-two-year-old housewife entered the hospital because of a mass in the right lung. One and a half years before admission the patient entered another hospital because of headache and weight loss. An area of density 3.0 by 2.5 cm. was visible by x-ray in the right lower lobe. It was smooth in outline and homogeneous in density. The lung fields were otherwise clear. There had been no cough or hemoptysis. Bronchoscopy was negative. The patient refused operation and was discharged. During the next year and a half she was constantly fatigued, but lost no more weight. The headaches became less frequent and severer. The attacks were often associated with dizziness and mild nausea. Mild exertional dyspnea appeared in the last six months. One month before admission a mild, hacking, nonproductive cough developed. There was no fever or sweating at night.

With the birth of a son thirteen years prior to admission, the patient had "milk legs," which kept her in bid nine weeks. Early in this period there was a sudden, sharp, pleuritic pain anteriorly in the left chest. This lasted a day and caused difficulty in breathing.

A cousin with whom the patient had close contact died of tuberculosis eight years before admission. At the age of twenty-four the patient had a bilateral salpingectomy. The pathological report was acute bilateral salpingitis. The periods thereafter were regular until admission, lasting from one to six days. The first day of the period was usually accompanied by cramping pains.

On physical examination there was a decrease in breath and spoken voice sounds over an area 4 cm. in diameter to the right of the spine over the right lower lobe. The heart was normal. There was a healed midline suprapublic scar, with an area of slight tenderness around it. The cervix was long and normal. The uterous was forward and attached to the abdominal wall, was larger than normal and felt round. A hard, firm, irregular mass was palpated along the right side of the fundus.

The temperature was 99°F., the pulse 72, and the respirations 20. The blood pressure was 110 systolic, 70 diastolic.

The red cell count was 4,180,000 with 12.7 gm. of hemoglobin. The white-cell count was 7,700, with 57 per cent neutrophils. The urine was normal. X-ray examination of the lung revealed a mass just below the right hilum, probably in the right lower lobe. It had not changed in appearance from the original film. A flat plate of the abdomen and an intravenous pyelogram showed normal urinary tracts. There was an ill-defined, irregular mass in the pelvis slightly more to the right than left measuring approximately 11 x 6 cm. in diameter. It did not appear to exert any pressure on the urinary tract. A barium enema was negative.

An operation was performed on the twelfth hospital day.

DR. ROBERT T. PHILLIPS:

This 32 year old housewife was admitted because of a mass in the right lung. Examination on admission revealed in addition to the pulmonary pathology, an irregular mass palpable along the right side of the uterine fundus. An operation was performed on the 12th hospital day.

In general consideration we have:

(1) No weight loss in the past 18 months
(2) No fever or night sweats
(3) Normal temperature, pulse respirations, blood pressure, blood count and urine

On these considerations I will omit discussion of acute or chronic infections processes and malignancies. In the first examination of the pulmonary lesion we have described an area density 3 by 2.5 cm in the right lower lung.

It was smooth in outline and homogeneous in density. The lung fields were otherwise clear. The patient had no cough or hemoptysis. Bronchoscopy was negative at the time of admission, 18 months later the patient had had mild dyspnea on exertion for six months. One month before admission a mild hacking non-productive cough developed.

On physical examination there was a decrease in breath and spoken voice sounds over an area 4 cm. in diameter to the right of the spine over the right lobe. X-ray examination of the lung revealed a mass just below the right hilum probably in the right lower lobe. It had not changed in appearance from the original film. The condition of the surrounding lung is not commented on.

So we have a mass in the right lung near the hilum. This mass is not inflammatory nor malignant I say because of the previously enumerated general considerations. The dyspnea and cough described would suggest that this process is slowly growing and has finally encroached upon the lumen of a bronchus. Further x-ray, bronchoscopic and lipiodol studies have been denied us. However, our patient is a woman and under forty.

Adenoma of the bronchus is a lesion occurring most frequently in women under the age of forty and in the right lower lung. It is a tumor of slow growth and gives few symptoms or findings until it encroaches on the lumen of a bronchus. Cough is the most frequent symptoms. The cough is usually non-productive early. An asthmaloid wheeze may be present. As occlusion of a bronchus occurs pulmonary suppuration with atelectasis pneumonitis, lung abscess and complications may develop. Treatment may be bronchoscoping or surgical removal of the area of lung involved. It would seem logical to assume that the first operation to be done on this patient would be bronchoscopic study and possibly removal or fulguration of the tumor if found.

As to the pelvic pathology. We are told that there is a hard firm irregular mass pulpated along the right side of the uterine fundus. The uterus was forward and attached to the abdominal wall. It was longer than normal and felt round. A barium enema was negative. An intravenous pyelogram showed normal urinary tracts with no evidence of pressure exerted by the pelvic mass.

Eight years previous to admission the patient had had a bilateral salpingectomy for acute bilateral salpingitis. The periods thereafter were normal. About the same time the patient had had close contact with a cousin who died of tuberculosis. So we have a woman aged 32 with a

mass along the right side of the uterus who eight years previously had bilateral salpingectomy.

As previously stated I have decided that this cannot be inflammatory or malignant. It seems most improbable but it is of course barely possible that this could be an ectopic pregnancy. However Sampson states that 74% of women subjected to salpingectomy develop endometriosis in the site of the tubal stumps. It is difficult to prove whether post operative endosalpingosis has resulted from an out growth of the mucosa from the tubal stump or bits of tubal or uterine epithelium transplanted into the wound during operation. There is also the possibility that mucosa transplanted into salpingectomy wounds may come from the uterine cavity. There is also the possibility that the ovary may become adherent to the salpingectomy wound and be invaded by endometrial tissue.

So—did the patient go to bronchoscopy or was a pelvic laparotomy done either way my diagnosis will be:

1. Adenoma of the bronchus
2. Endometriosis of the right tubal stump.

DIFFERENTIAL DIAGNOSIS

Dr. Donald S. King: As Dr. Schatzki said in the preceding case we are constantly faced with the problem of what to do with cases that show in the x-ray film a small rounded area of density in the lung field. Within the last three weeks we have seen such shadows in three cases similar to the one that is presented to me today. I should like to put up the films on these three cases along with the film of the case that I am to discuss. The first film is the film of today's case. Unfortunately this is only a Bucky film which does not give the same density as an ordinary film. The lateral film in this case is also shown. As one notes, the three other cases are different from the case under discussion in that the lesions are in the peripheral area of the lungs and not near the hilum. The first of the outside patients had a negative x-ray film in 1939. A film taken in 1942 showed a rounded lesion. Fortunately I was away at the time this film was taken and did not have to decide what was to be done about it. We have other films taken in 1944 and 1946 which show that the lesion had changed very little if at all. The second case was one in which the lesion was picked up by routine exam of all employees in a large Boston Department Store; no symptoms of pulmonary disease. In the third case there were definite pulmonary symptoms and the decision to operate will probably not be difficult.

In the discussion of today's case we noted first the story of pleuritis pain which lasted a day and caused difficulty in breathing. There seems little question that the symptoms were due to pulmonary infarct. I do not believe there was any relation between this episode and the rounded lesion which appeared some years later.

As in the case discussed by Dr. Schatzki, this patient was also exposed to tuberculosis, and the diagnosis of tuberculosis must therefore be considered. There is a possibility that the bilateral salpingitis was also of tuberculous nature, but I assume that the pathological report of acute salpingitis rules out tuberculosis.

There was a decrease in breath and spoken voice sounds over an area 4 cm. in diameter to the right of the spine. This examiner certainly made a careful examination. I shall wager anything that he had seen the film before he made that report.

I assume from the description of the pelvic examination that there were two masses one of which was the uterus itself and the other outside the uterus, but I cannot be absolutely sure of this from the description.

The most important sentence in the whole history of the case is in the x-ray report which reads "it had not changed in appearance from the original film." We are therefore dealing with a lesion that did not change in 1½ years. The x-ray department goes on to report. "An intravenous pyelogram showed normal urinary tracts." Obviously the doctors in charge of the case were looking for evidence of a hypernephroma from which there had been a solitary metastases to the lung.

I suppose again, as in Dr. Schatzki's case, that the operation performed was an operation on the lung and not on the pelvis and we are again faced with the question of the nature of the round shadow. My first inclination was to bet again on tuberculosis because of the story of exposure and because the symptoms were somewhat suggestive of tuberculous infection. But there are two things against this diagnosis. In

"A high percentage of cases of seasickness and carsickness can be aborted or prevented by suitable doses of dimenhydrinate (Dramamine)."

—Council on Pharmacy and Chemistry, New and Nonofficial Remedies, J.A.M.A. 143:815 (July 1) 1950.

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RESEARCH IN THE SERVICE OF MEDICINE **SEARLE**

the first place, with a small rounded shadow due to tuberculosis there is evidence of tuberculosis elsewhere in the lungs. This is however not necessarily true. In the second place, except in a calcified primary complex the rounded shadow due to tuberculosis or so called tuberculoma, usually change in appearance in 18 months' time. I believe almost all of the tuberculomas that we have observed have changed in the x-ray film in that length of time. I am therefore inclined to say that the lesion under discussion is not tuberculous.

As I noted in the beginning this rounded shadow is near the hilum where we have seen some bronchiogenic cysts and some of the smaller cysts in this area have given somewhat similar x-ray appearance. Usually, however, the area of density is larger and gives a somewhat different shadow. I do not believe that this picture is sufficiently characteristic of a bronchiogenic cyst to warrant such a diagnosis.

Primary carcinoma of the bronchus and metastatic cancer are both, I believe, ruled out by the fact that there was no change in the film in 1½ years. So far as our experience goes I do not believe there has been a single case of primary or metastatic cancer of the lungs in which the x-ray shadow has not changed in this length of time. Even the hypernephromas, some of which are very slow growing have changed in that length of time.

Of course I am concerned about the condition in the pelvis. As I went over the case I was about to forget that there was anything wrong there and until the discussion of Dr. Schatzki's case I had not given a thought to the possibility of a pulmonary metastasis from the pelvis. Assuming that the operation was on the lung it is quite obvious that the doctors in charge did not believe there was a pelvic cancer from which a metastasis might occur.

Dr. Tracy B. Mallory: You are wrong about that. The operation was on the pelvis.

Dr. King: That is, of course, a bombshell. I was going to forget about the pelvic disease so I am even more interested in what you think about the pelvic condition, Dr. Meigs.

Dr. Meigs: I have a note on the record that I shall try to find.

Dr. Fletcher H. Colby: I am not convinced that metastatic lesions in the lungs necessarily change as you say in a brief period.

Dr. King: The only ones I can be sure about are the ones I have followed personally and all of these increased in size in a few months. In the famous case of hypernephroma in which the kidney was first removed and the metastasis taken out several months later with apparent cure after 12 years, the metastatic lesion had almost doubled in size in a year's time. We have also recently followed a case at the Baker Memorial Hospital in which the hypernephroma was taken out five years ago and metastatic lesions appeared in the lung one year ago. The lesions in the lung have unmistakably increased in size during a years observation. What the films might have shown had they been taken in the 4 year period before the metastatic lesions were discovered I do not know.

Dr. Colby: Some are fairly slow in growing. They must be because secondary deposists may appear even as late as 15 years or more.

Dr. Meigs: I shall read part of my note. "The cervix is long and normal in appearance. The uterus is forward and attached to the abdominal wall; it is large and round, definitely larger than it should be. The left side is negative but in the right side I believe I feel a hard, firm irregular mass. This mass probably represents the right ovary, although with tumor in the lung I think we ought to consider the question of neoplasm. Dr. Benedict should be asked if he would like to peritoneoscope her. If he does not and all studies on your service are negative we would be very glad to explore her."

The question is, what kind of mass is it? Apparently she has no abnormal menstrual periods. One would have to consider the possibility of a fibroid growing off the side of the uterus. It might be in the form of leiomyosarcoma which is a malignant tumor and may metastasize to the lung. Pelvic inflammatory disease we rule out because she had both tubes removed. One would have to consider ovarian tumor and it could even be endometrial cancer.

Dr. King: We do not know what this lesion in the lung was then?

Dr. Mallory: Yes we do.

Dr. King: And you know what the pelvis showed too?

Dr. Mallory: Yes.

Dr. King: We are now faced with the question whether we can connect the two processes. I came determined not to consider the pelvis as a possible source of metastasis and I shall keep to the line of approach. I do not believe that this is metastatic cancer.

I have already discarded tuberculosis and bronchiogenic cyst. The question raised in the previous case about benign adenoma interests me a good deal. Until 1942, except for one possible case all the adenomas we had followed were at least in part in the lumens of the larger bronchi and could be definitely proved by bronchoscopic biopsy. I was told that while I was away there was a case admitted from another hospital in which operation had shown an adenoma that was entirely outside the bronchus. The case under discussion may be that case. In view of the fact that this lesion has not changed in x-ray appearance in 1½ years and that it is close to the large bronchi, adenoma must be considered as a possible diagnosis.

There are a multitude of other benign tumors that should be mentioned, but it would be pure guess-work to try to name any other tumor. Fibroma, chondroma and hamartoma are all possible but taking everything into consideration I should say adenoma.

CLINICAL DIAGNOSIS: Adenoma of bronchus.

Dr. King's Diagnosis: Benign adenoma of lung.

ANATOMICAL DIAGNOSIS: Adenoma of bronchus. Endometriosis of ovary.

PATHOLOGICAL DISCUSSION:

Dr. Mallory: The first operation was on the pelvis and an ovarian mass was found to be due to extensive endometriosis of the ovary. About 10 days later the chest was explored and Dr. Sweet will tell you about that.

Dr. Sweet: This patient was admitted on the Gyn. Service and I was asked to see her before the pelvic examination relative to what was wrong in the chest. I thought we should see what was wrong in the pelvis first because at that time we did give some thought to metastatic tumor. Having proved that the pelvic tumor was benign and therefore not responsible for the pulmonary lesion we did go ahead with the chest operation. I came to the same conclusion as Dr. King—that it was an adenoma. It was an interesting operation because when I first exposed the tumor it was in the lower lobe, presenting on the fissure as it lay against the middle lobe. I thought that I could excise the tumor locally. There was not much lung tissue over it. I believed it was something I could pry out without difficulty but in dissecting I saw that it was actually growing out of a bronchus, and I did a lobectomy. The further pathological details we shall hear from Dr. Mallory, but I was convinced at the time that I was taking out an adenoma.

Dr. King: Was it inside or out?

Dr. Sweet: Both.

Dr. Mallory: I have the gross picture of the resected specimen. You can see the tumor without any difficulty almost completely circumscribed, but it was definitely adherent to the outer portion of the bronchial wall. There was no involvement of the bronchial mucosa and no narrowing of the bronchus by tumor. These tumors ordinarily arise within the bronchus although they may also extend outward between the cartilages and form a dumbbell shaped mass. This is a unique case, in our series at any rate, in that the tumor lay entirely outside the bronchus with no projection in the bronchial lumen.

Dr. Meigs: This patient had bilateral salpingitis and endometritis 8 years before admission. It is rare to see endometriosis under such conditions when the tubes are closed. This seems to refute Sampson's theory of menstrual blood flowing back through the tubes and producing endometriosis. If she had had the condition when she was first operated on it would probably have been noted.

Dr. Mallory: You might have noted it but most surgeons do not see it as readily as you do.

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TOPICS OF CURRENT MEDICAL INTEREST

RX, DX, AND DRS.

By GUILLERMO OSLER, M.D.

For those who missed it at the A.M.A. meeting, Hench and colleagues have reported an item which seems ahead of its time. Cortisone and Compound F are *absorbed and effective by mouth* . . . The hormones are still in scarce supply—but when they increase to the 'pound and penny' stage, the knowledge can be used.

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A previous paragraph or two about versatile doctors has prepared the way for this one about Dr. Ward . . . Dr. J. P. Ward, that is. Our own Health Dept. Chief . . . We didn't allow for the width of versatility, though. Dr. Ward is the *winner of a cartoon caption contest* in a medical advertising mag! . . . This mention of it is not ridicule, just envy.

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Research in Arizona could well repeat and expand on a piece of Swedish investigation, published two years ago in the Annals of Rheumatic Disease . . . Drs. Edstrom, Lundin, and Wrammer reported on *Hot Climate for Arthritis*, or, 'Investigations into the effect of hot, dry microclimate on peripheral circulation, etc., in arthritic patients' . . . Thirty-four patients with rheumatic fever, and others with rheumatoid arthritis, were kept in an air-conditioned room for 100 days. The temperature was 89 F., the humidity 35% . . . Symptoms decreased, the peripheral vascular bed dilates widely, vascular reflexes decrease, oxygen saturation of venous blood (low in arthritis) rises above normal, and beta hemolytic streptococci are usually eliminated from the throat . . . Southern Arizona should be able to put into figures what we suspect the 'macro' climate does.

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No luck yet in finding out the results of studies on *desert warfare* . . . It was said that the Army had some interesting data during the last war, and it seemed that it might be applied to making living and therapy more satisfactory in the Southwest . . . The U.S.P.H.S. doesn't know of it, and it might not be wise to ask the Army about it right now.

Dr. Perrin Long of Johns Hopkins is one of the pioneers of chemo and antibiotic therapy. Every now and then he gives a summary of the use of the best agents, and he has done it again . . . The Pennsylvania Medical Journal contained *a comparison of penicillin, streptomycin, aureomycin, and chloromycetin* in a recent issue, and it was abstracted in 'Modern Medicine' . . . We have written to see if the charts can be reprinted in ARIZONA MEDICINE. They would be very valuable to have, and can even be further modernized.

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Histoplasmosis may occur in a progressive form. It is rare but is usually fatal . . . There has been no therapy for most fungus infections, and none for histoplasmosis until a report by Christie of Vanderbilt . . . He used a wartime fungicide called *ethyl vanillate* in treatment of five children, with recovery in all.

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The *Blue Cross plan* sometimes takes a beating. Everyone has read Dr. Paul Hawley's plea that physicians help to protect the plan. Needless hospitalizations and useless charges can be cut out by the careful doctor, and the costs and losses can be reduced . . . We agree, we condemn waste. We urge honesty and thoughtful use of the Blue Cross privileges . . . But (and here is where the shoe personally pinches) we have a feeling of sympathy for the *tuberculous patient* whose income has been cut off because his policy has been shifted from a 'group' to a 'direct' payment contract. It hits the tuberculosis hospitals where they live, too.

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A former Arizona Dept. of Health worker has moved up in the Federal service. *Dr. Arnold B. Kurlander*, on loan here before his three year service in Ohio, has been transferred to similar work in the Division of Tuberculosis at Washington, D. C.

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We'd give a nickel to know whether 'Banthine,' a brand of methantheline bromide, is of any more value than most approaches to *peptic ulcer therapy* . . . It acts as a parasympathetic blocking

agent, decreasing gastric secretion and intestinal motility. It is anticholinergic, like atropine, but perhaps is more selective . . . Pain has been found to be markedly reduced, the heart effects are scanty, but the degree of action on the eyes and salivary glands is not yet certain . . .

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The risk of cataract formation from gamma and roentgen rays and from neutrons is said to be lessened by a 50% improvement in *protective glass* . . . The glass can be used for eye-glasses, or for peepholes in safety barriers of atomic reactors . . . The two kinds of glass were developed by five chemists at the University of Pittsburgh. The X and gamma ray glass contains tungsten phosphate. The neutron-stopper contains cadmium borosilicates and fluorides . . . Four feet of earth vs eight inches of cement vs a few cm. of glass.

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One of the finest summaries which has come to this desk-in-the-desert is that of Armour and Company on 'ACTH.' Very clear, comprehensive and up-to-date—plus one additional rare quality—the presence of a *price list* which is so often missing when drugs or equipment is being discussed.

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The lead article in the July 29th JAMA was by Gordon and Albright . . . Their authority in writing of the *treatment of thyrotoxicosis* by radioactive iodine can be judged by the sentence "The present report deals with the first 120 cases treated—". . . This is the same Dr. Edgar Gordon of Wisconsin who gave ARIZONA MEDICINE such a beautiful case-analysis in January, 1949, with its ultra-modern treatment of hypothyroidism and hypoparathyroidism.

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The *history of pneumonectomy* is an amazing one. First, because it is only of 19 years duration and, second, because it progressed so regularly and well . . . Rudolph Nissen tells of removing the first entire lung in Sauerbruch's German Clinic in 1931; less than two years later Cameron Haight of Michigan removed one for bronchiectasis; shortly afterwards Evarts Graham of St. Louis removed one for carcinoma; and a few months later Rienhoff of Baltimore developed the currently used 'individual ligation' technique . . . The final amazing part of the story is that Nissen's patient was alive when last located, Haight's is married and has a child, and Graham's is now a practicing physician, aged 48 years.

Arizona chest surgeons have done a fairly high percentage of the *lung resections for coccidiomycosis*, alias 'valley' or 'desert fever' . . . Melick of Phoenix has summarized the cases by correspondence with the members of the A.A. for Thoracic Surgery, for publication in that society's journal for July 1950 . . . Replies were received from 224 individuals in 37 states, Canada, Sweden, and Hawaii. One hundred and nine cases were reported by 39 surgeons or groups . . . Melick had done 8 resections, Howell Randolph of Phoenix had done two, and Stacey of Tucson had done one . . . (We also have more than our share of the disease.)

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It was certain that *anti-coagulant drugs* would be improved, and some of the early efforts are now available . . . A drug in the coumarin series is Tromexan, and it acts more rapidly and more briefly on the prothrombin time than Dicumarol . . . A drug with a similar action to heparin is called Paritol . . . Decision on the relative effects will have to wait, as usual.

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A *new transfusion method* has been described by Dr. Carl Walter of the P. B. Brigham Hospital in Boston . . . Dr. Walter's equipment seeks to provide a donor set which (a) avoids a vacuum, (b) does not make use of glass, and (c) utilizes expendible materials . . . The blood is collected into a *transparent plastic bag* through plastic tubing. Absorptive resins in the tube remove the calcium and magnesium in the blood, and remove the need for anti-coagulants. The bag does not contain air, and the blood flows into it by venous pressure . . . It flows out of the bag either by elevation, squeezing by hand, or by the weight of the body as the patient lies on it.

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In a pre-meeting summary of A.M.A. topics, Dr. M. J. Whitelaw of Phoenix was not credited with two papers, 'The Treatment of Juvenile Acne with Topical Estrogens,' and 'The Influence of Female Hormones on the Basal Body Temperature.' He also presented a film called 'The Use of the Cervical Cap in Oligospermia,' and discussed a paper 'On Sterility in the Male' . . . He did *not* discuss a paper on prostatic massage, as promised in the A.M.A. handouts, and repeated here.

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Already *the alcoholic* has a way to circumvent the 'autabuse' treatment . . . Even if someone

forces him to take his pills regularly, all he has to do is to carry the equipment, a solution of ascorbic acid, ferrous chloride, and distilled water, and give himself an intravenous injection before he takes a drink, or later when the nausea reaction occurs . . . If this suggestion seems exaggerated, just think back on the cute and complex and fantastic things an alcoholic does to *get* a drink . . . Jokivarto of Finland says, in the Quart. J. Studies on Alcohol, that it works.

—♥—

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PERSONAL NOTES

Dr. Albert Eckstein, Phoenix, was certified by the American Board of Internal Medicine, June, 1950.

Dr. Douglas D. Gain, Phoenix, has returned to practice of radiology after a year's post-graduate study in Philadelphia, Pennsylvania at the Graduate School of Medicine, the University of Pennsylvania.

He has been appointed head of the Department of Radiology at St. Monica's Hospital, Phoenix. Dr. Gain was certified by the American Board of Radiology in 1950.

Dr. L. D. Beck, Phoenix, attended the Oklahoma State Medical Convention, June 4-6th at Oklahoma City.

Dr. R. A. Gutekunst, Phoenix, has resigned as Resident Physician at the State Tuberculosis Sanitorium at Tempe. He has taken a position at Torrance, California at the Harber General Hospital.

Dr. Evelyn B. Johnson, Minneapolis, Minnesota, has come to the Maricopa County Health Unit as Pediatrician. She formerly was Pediatrician and Associate Cardiologist with Minneapolis Children's Heart Clinic.

Dr. Clarence G. Salisbury, Ganado, has accepted appointment effective September 1st, as Chief of the Bureau of Preventive Medical Services, Arizona State Department of Health.

Dr. Richard B. Johns, Phoenix, has accepted a Residency at Children's Hospital, Denver, Colorado.

Dr. James Ovens, Phoenix, completed a years residency in cancer surgery at the M. D. Anderson Hospital, Houston, Texas.

Dr. E. Payne Palmer, Sr. and *Dr. Joseph Bank,* Phoenix, are attending the International Conference on Cancer, in Paris, France.

Dr. Archie Cruthirds, Phoenix, attended the Sectional Meeting of Scottish Opthalmological Society, Glasgow-Edinburgh, Scotland, July 10th-14th and the International Congress of Opthalmologists, London, July 17th-21st, and the International Congress of Surgery, Argentine Chapter, Buenos Aires, Argentine, August 1st-8th.

Dr. Kenneth C. Baker, Tucson, attended the meeting of the Pacific Coast Dermatological Association in Oakland, California, September 1st to 4th inclusive.

NOTICE

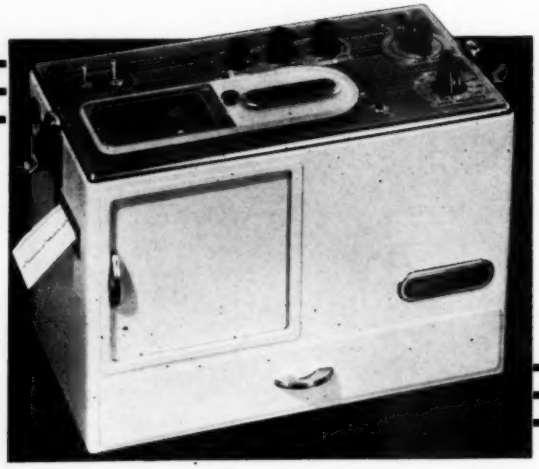
The Fifteenth Annual Assembly of the United States Chapter of the International College of Surgeons will be held in Cleveland, Ohio, October 31 to November 3, with headquarters at the Cleveland Hotel.

Surgical clinics will be held in several Cleveland hospitals on Monday, October 30. All scientific sessions will be held at the Cleveland Public Auditorium 9:00 a.m. to 5:00 p.m. Tuesday through Friday. A most excellent program has been arranged at which time some of the most prominent surgeons of America, and some foreign speakers, will discuss the current contemporary surgical scene.

Through the courtesy of Smith, Kline and French Laboratories, a fine colored television program of surgical procedures, originating from the St. Vincent's Charity Hospital, Cleveland, will be shown daily in the auditorium from 9:00 a.m. to 1:00 p.m. Motion pictures will also be presented each day depicting many of the recent advances in surgery and surgical technique.

One of the highlights of the meeting will be the annual banquet at the Statler Hotel on Thursday evening when America's great surgeon, Dr. Frank Lahey of Boston, will talk on "Some of the Recent Advances in Surgery." Dr. Elmer Henderson, President of the American Medical Association, will deliver an address on "The Importance of International Cooperation in Surgery."

Reservations may be secured by writing to the Committee on Hotels, International College of Surgeons, 511 Terminal Tower, Cleveland 13, Ohio. Preliminary programs may be obtained from the central office, 1516 Lake Shore Drive, Chicago 10.



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ANNOUNCEMENT OF VAN METER PRIZE AWARD

BY GEORGE C. SHIVERS

The American Goiter Association again offers the Van Meter Prize Award of Three Hundred Dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The Award will be made at the annual meeting of the Association which will be held in Columbus, Ohio, May 24, 25 and 26, 1951, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length; must be presented in English; and a typewritten double spaced copy in duplicate sent to the Corresponding Secretary, Dr. George C. Shivers, 100 East Saint Vrain Street, Colorado Springs, Colorado, not later than March 1, 1951. The committee who will review the manuscripts is composed of men well qualified to judge the merits of the competing essays.

A place will be reserved on the program of the annual meeting for presentation of the Prize Award Essay by the author, if it is possible for him to attend. The essay will be published in the annual Proceedings of the Association.

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SECRETARY'S LETTER

Philip R. Overton, general counsel for the State Medical Association of Texas, wrote an interesting letter recently to a physician in a Texas veterans hospital relative to membership dues in the American Medical Association. Two paragraphs of Mr. Overton's letter follow:

"Since I am not a doctor, but an attorney for the State Medical Association of Texas, it is necessary that I look upon your questions relative to dues in a personal way and with that in mind I feel that any individual who has prepared himself to practice a profession, such as medicine, should be willing to pay the $25 dues to his national organization. It would be a matter of pride with me as an individual and certainly I would want to carry my part of the load in defeating the socialistic trend now being fostered in this country; and too, while you or others in government service might not wish to become involved in political considerations as an American citizen it is my opinion that it would be your duty to express your opinion even though it might be in disagreement with that of your own state and national associations, and to do everything possible to see that the principles on which this country was founded are maintained. To my mind, there has been too much complacency, not only among the medical profession, but among the lawyers and the other professions as well as the people generally.

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BOOK REVIEW

PRACTICE OF MEDICINE. By Jonathan Campbell Meakins, C.B.E., M.D., LL.D., D.Sc. Fifth Edition. Pp. 1558. Illustrations 518 including 50 in color. The C. V. Mosby Co., St. Louis. 1950.

In the preface to this fifth edition the author begins by saying, "The task of writing a new edition of THE PRACTICE OF MEDICINE is becoming increasingly difficult . . . medicine is being clouded by a multiplicity of minutiae which are beyond the technical capacity or facilities of the general physician." When one reflects on this statement and gains some comprehension of the task of weeding out all of the non-essential minutiae which would fill many volumes and setting down the essentials in only one manageable volume, he is inclined to be tolerant and overlook any shortcomings or omissions as inevitable. Dr. Meakins has done an excellent job with a minimum of these shortcomings.

The arrangement of the text in chapters dealing with disease as related to the various regions and functional systems of the body has several obvious advantages. It makes it easy to locate the reference to any specific disease and this is further facilitated by a very good index. The index itself occupies fifty-five pages. The arrangement also allows an introduction to each chapter giving the reader a brief review of the physiology and pathology of the region or system and also a discussion of the symptomalogy of the system. Correlation of similar diseases is effectively done under this arrangement. These features should be very valuable to the student and make the book useful as a quick reference work for both the specialist and the general practitioner.

The descriptions of diseases are comparatively brief but are clear and concise. The specialist will find many omissions but most of these can be classified as non-essential minutiae. The specialist must go to his specialty references for these. The illustrations are for the most part of high quality and good selection. A few of the colored ones appear to have been carried over from earlier methods of color reproduction but are satisfactory. The color plates of blood smears and bone marrow smears as found in the chapter on diseases of the hematopoietic system are particularly good. Incidentally the text of this chapter is also very well written and very much up to date.

A chapter on Chemotherapy and Antibiotics has been added. Several chapters have been rewritten. One omission which may be considered unpardonable has been noted. That is the omission of any reference to unipolar electrocardiographic leads in the diagnosis of coronary artery disease. In this reviewer's opinion differential diagnosis has not been given the attention it deserves in a book which will be used by medical students but perhaps this has been compensated for in the excellent treatment of symptomalogy and positive diagnosis.

In conclusion I would say that Dr. Meakins has provided a very valuable reference work on general medicine which is surprisingly up to date in this time of rapid change in medicine. It should be very useful not only to the student and general practitioner, for whom it was probably primarily intended, but to the specialist as well.

R.L.F.

BREAST DEFORMITIES AND THEIR REPAIR. By Jacques W. Maliniac, M.D. Cloth $10. P. O. 193 with numerous illustrations and diagnosis. Greene and Stratton, Inc. 381 Fourth Avenue, New York 16, N. Y. 1950.

This concise booklet presents in a small volume the history, anatomy and physiology, and the surgical treatment of all types of breast deformities. Brief descriptions of operative procedures long abandoned are given showing the reasons why they were given up, as a prelude to the more thorough discussions of plastic procedures now in use. To the plastic surgeon called upon to perform mammary plastics this will be an excellent reference all in one source, while to the general surgeon occasionally performing or contemplating performing plastic procedures on the breast this will be a valuable aid in avoiding some of the pitfalls encountered in the early surgery in this field.

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MONTANA DOESN'T LIKE SOCIALIZED MEDICINE

When George B. Larson of the A.M.A. Bureau of Exhibits, drove to the Montana State Medical Association meeting in Bozeman recently, he was stopped at the state line by a husky greeter, dressed in a flaming red shirt, overalls and western sombrero.

"Welcome, pa'dner," said the man with a smile, and handed Larson a big envelope from the Montana Chamber of Commerce. All out-of-state cars are stopped at the border and every driver is given one of the envelopes containing literature extolling the beauty spots of the state.

Mr. Larson thumbed through the material later and, much to his amazement, found two mimeographed papers carrying copies of resolutions passed by the Montana Chamber of Commerce against socialized medicine.

One resolution said: "We oppose federal medicine for the simple reason that like every other key plank to Marxian socialist doctrine it simply will not work. No nation in the world has better medical care or is in better health than the United States. This obvious fact leads us to believe that socialized medicine is a scheme to exploit the doctors who are a minority in order to pretend to distribute benefits to the voting majority who are the people. This is dishonest."

Judging from Mr. Larson's experience, it is very evident that the hard work done by Montana doctors against socialized medicine is paying off. Congratulations!

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REPORT

Council on the Management and Treatment of Diseases of the Chest,

American College of Chest Physicians

Considerable attention has recently been given to BCG vaccination in the public press. From this publicity the impression might be gained that this procedure alone holds promise of real control of tuberculosis. Since such an impression might postpone indefinitely the establishment and extension of accepted control measures, this statement of the status of vaccination in tuberculosis control programs is issued.

1. Control measures in tuberculosis should be directed at eradication of the disease as a major cause of death or disability.

2. The marked improvement in tuberculosis mortality figures, particularly for the ages under 30, demonstrates the effectiveness of the present control program.

3. The low rate in children and the continuing high rates in adults over 50 emphasize the location of the problem at the older age levels rather than in children. Under these circumstances, the efficiency of a method of tuberculosis control would be measured by its effect on the mortality from tuberculosis in the older age group, rather than in children.

4. The addition of a vaccine to the present control program requires both careful and adequate consideration. Of the vaccines proposed, BCG has been used most widely and is the one most often discussed.

5. This has been used for more than 25 years and recently many millions of people have been vaccinated. However, it must be stated that there is no evidence that meets strict scientific requirements demonstrating that BCG affects the control of tuberculosis, despite the very suggestive results of a few studies.

6. Because of the above fact and because there is no general agreement among investigators anywhere in the world on such fundamental matters as the preparation of vaccine, the method of vaccination, what constitutes a successful vaccination, how resulting immunity may be measured, how long such immunity lasts, etc., the procedure would seem to be still in the investigational period.

7. It is therefore recommended that investigation of vaccination in tuberculosis be continued and increased under standard and stringently controlled conditions. This investigation should be designed to determine if the vaccine is indeed effective and what the limitations of its use might be. It would seem desirable that in each country, one agency preferably the official health agency, should have control of the investigation.

8. Until this has been determined and until these controlled studies are completed, the use of BCG vaccine should be limited to such investigative studies.

9. At the present time the methods which have been proved effective in tuberculosis control should be increasingly applied to all segments of the population, regardless of decreasing mortality figures, so long as tuberculosis remains an important cause of death. These measures include mass x-ray case finding, early diagnosis, rapid institution of treatment, isolation of open cases, and the restoration of the patient to normal life.

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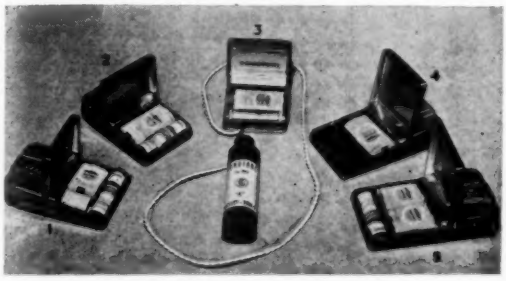

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BRITISH DOCTORS WANT TO QUIT HEALTH SERVICE

British doctors are becoming more and more fed up with red tape under the British National Health Act.

Last week newspapers reported that delegates to the annual meeting of the British Medical Association adopted a resolution urging preparations for a withdrawal from the system because of delay in negotiations on payment of general practitioners.

General practitioners now are paid from a pool calculated at a yearly rate of $2.52 a head of 95 per cent of the population, known as a capitation fee.

For the current year this pool is about $112,000,000 and the amount paid to each doctor depends on the number of patients he has under the system.

The doctors contend that the pool should be increased by $46,200,000 a year from that of July, 1948, when the scheme began, and that the capitation fee should not be fixed but be on a sliding scale.

One delegate told newsmen that doctors had been turned into form-filling drudges, that the health service was in a mess and that it was getting worse.

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